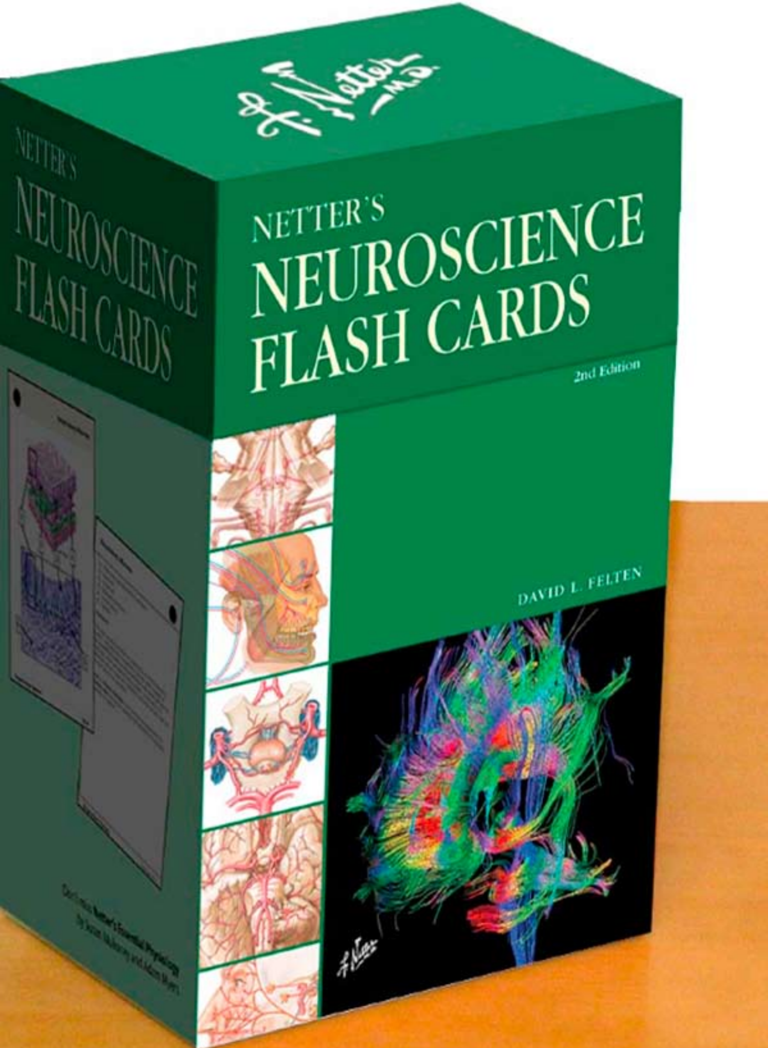

NETTER'S
NEUROSCIENCE FLASH CARDS

2nd Edition

DAVID L. FELTEN

F. Netter M.D.
NETTER'S
NEUROSCIENCE
FLASH CARDS
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Netter's

Neuroscience Flash Cards

Second Edition

David L. Felten, MD, PhD

Acknowledgments

Netter's Neuroscience Flash Cards, second edition, is a logical follow-up to *Netter's Atlas of Neuroscience*, second edition. Both publications are intended to provide a visually oriented, succinct roadmap to the basic neurosciences and their application to human disease and its treatment. I have thoroughly enjoyed working with Julie Goolsby (Associate Developmental Editor), Marybeth Thiel (Senior Developmental Editor), and Elyse O'Grady (Editor, Netter Products) from Elsevier in the long and challenging process of producing the *Flash Cards* and *Atlas*. Their helpfulness, diligence, professionalism, and commitment to bringing the Netter illustrations to yet more generations of physicians and health care professionals are inspirational. I also gratefully acknowledge the mentorship and wonderful teaching of Institute Professor Walle J.H. Nauta, M.D., Ph.D. from the Massachusetts Institute of Technology; his superb organization of the nervous system, brilliant insights, and use of overviews has inspired generations of neurosciences researchers, educators, and practitioners. I gratefully acknowledge the love, support, and encouragement of my wife, Dr. Mary Maida, who continues to tolerate mountains of books and papers piled all over the house and office while I work on my Netter projects.

And finally, I acknowledge the inspiration that my mother, Jane E. Felten, provided through her long challenge with the aftermath of polio. Although she was badly crippled by the disease at the age of 8, she never let this neurological disease interfere with a full and happy life. Her example demonstrates that the strength of human determination, will power, and faith can be powerful motivators for successful living, and that the indomitable human spirit can overcome even the most daunting challenges. Jane Felten demonstrated one of the fundamental principles of medicine- that one can be truly healed even in the face of severe pathology and disease, and in the absence of a "cure." She fought the good fight and lived a rich and rewarding life for more than 70 years. This project is dedicated to her memory.

SAUNDERS
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Preface

Netter's Neuroscience Flash Cards consists of selected illustrations from *Netter's Atlas of Neuroscience*, second edition, published in 2009. Reference to the corresponding figure number in the *Atlas* can be found on the front of each card. Relevant structures are labeled on the front of each flash card illustration with A, B, C, and so forth. On the back of the flash card is a list of all labeled structures. In some instances, such as the illustration of the cranial nerves, the schematic of hypothalamic nuclei, or the limbic forebrain structures, the labels are comprehensive enough to include all of the major components, providing the student an opportunity to test his/her knowledge in a more thorough fashion than with just a few labels.

In addition to the list of labeled structures, the back of the flash card also includes a comment. The comments consist of two types of information: (1) organizational information about the illustration that provides a summary of the structure or system whose components are labeled on the front (e.g., a brief summary of visual system projections and their functional role on the flash card demonstrating the extent of retinal projections); (2) brief discussion of points of clinical relevance to the illustration on the front of the flash card. Approximately half of the flash cards have comments that address the former, and half have clinical comments. The choice of comments on the back was determined by the author's consideration of what type of information would be most useful to a student trying to master the challenging field of neuroscience. *Netter's Atlas of Neuroscience,* second edition, contains extensive clinical comments, in contrast to the first edition. For the more detailed clinical points, the *Atlas* itself is the appropriate source.

Students have the delightful habit of asking challenging questions related to illustrations—such as those used in *Netter's Atlas of Neuroscience*, second edition—and include, "So how does this fit in with big picture of how the brain works?" or "So why do I need to know this information?" As information resources continue to expand, the ability of students to become more selective in their time allocation, to review helpful summaries of information that are still thorough and accurate, to succinctly learn about the clinical relevance of information to patient care, becomes more and more valuable. These flash cards provide assistance toward that end. However, I also remind my students that they need to know the underlying basic science principles and mechanisms so that the clinical relevance is a logical follow-up, not just a bunch more facts to memorize.

In preparing the flash cards for the second edition, I also took into account the wonderful new technology that enables the student to obtain ipod or iphone downloads, useful both for studying or exam preparation, and for reviewing information of relevance to a specific patient with a neurological problem or symptom. I tried to provide comprehensive enough information in the labeling, as well as a useful succinct summary, to allow the student to review or refresh information that can be useful in the consideration of a patient's diagnosis or evaluation.

The study of neuroscience ultimately requires that the student must develop an understanding of the application of scientific knowledge to human illness and pathology. In medical school courses, clinical correlations and the use of clinical examples are used to assist students to find the bridge between basic sciences and clinical application. *Netter's Atlas of Neuroscience*, second edition, and *Netter's Neuroscience Flash Card*s, second edition, have been written to optimize this process. Most students do not want to wallow through a 1,500 page textbook looking for relevant material buried in a seemingly endless discourse on esoterica. Until the student develops a solid "overview" of the field, and is able to place structures, pathways, systems, symptoms, and neurological phenomena into proper context, the large references will be confusing and more trouble than they are worth. Most students want to "cut to the chase" and only later will they seek more detailed information when it becomes important for the care of a patient. These *Flash Cards* are designed to "cut to the chase."

During my childhood, the use of flash cards was an enjoyable way to learn essential information and approaches. It is in this spirit that *Netter's Neuroscience Flash Cards* were developed. Students who have the persistence and drive to use these *Flash Cards* to review fundamental neuroscience with a strong slant towards succinct organization and clinical relevance will emerge with a surprisingly broad array of knowledge about major neurological applications of the basic sciences. They also will find themselves unusually well prepared for licensure and board certification examinations that require such knowledge. I wish the users of these *Flash Cards* and *Netter's Atlas of Neuroscience*, second edition, both success and enjoyment as they follow this visually-oriented learning format, and have the opportunity to benefit from the wonderful illustrations and medical knowledge of Dr. Frank Netter. It is the success of my students that makes the endeavor of teaching truly worthwhile.

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1 Overview of the Nervous System

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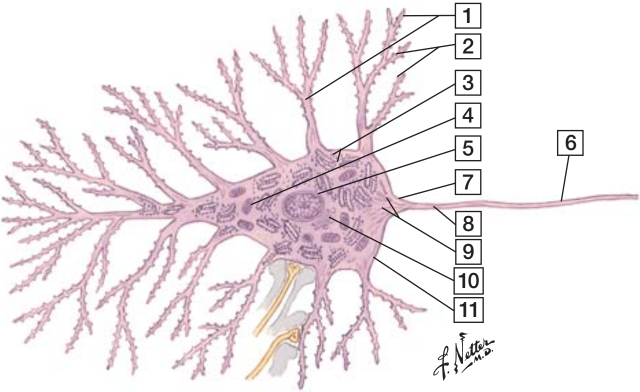
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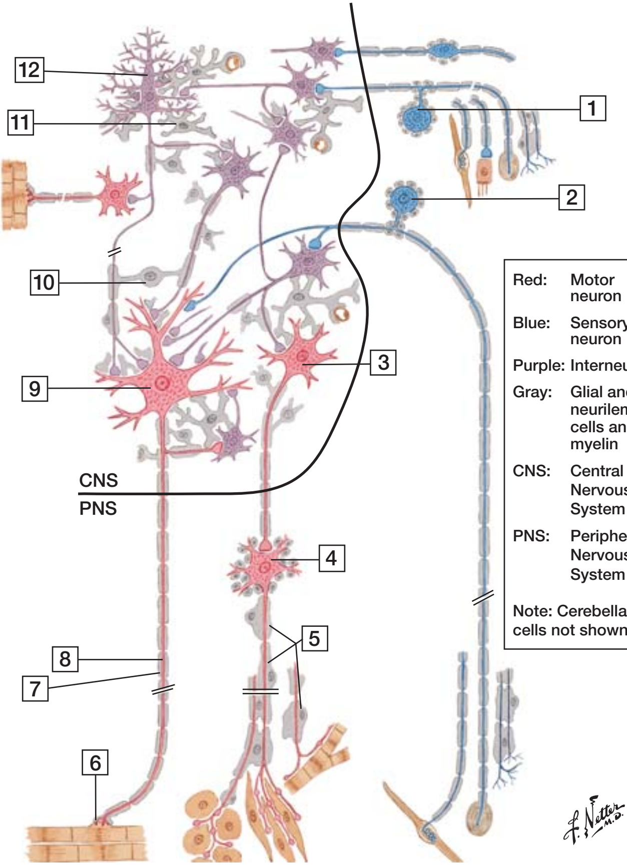
F. Netter M.D.

Neuronal Structure

1. Dendrites
2. Dendritic spines
3. Rough endoplasmic reticulum
4. Mitochondria
5. Nucleus
6. Axon
7. Axon hillock
8. Initial segment of axon
9. Neurotubules
10. Golgi apparatus
11. Cell body (soma)

Comment: The neuron is a highly active cell metabolically, with great demands for oxygen and glucose related to aerobic metabolism, especially important for maintaining ion gradients across the neuronal cell membrane. Neurons have very little metabolic reserve and depend on moment-to-moment delivery of oxygen and glucose. Neuronal form is related to the function of each individual neuron. Dendritic arborizations reflect the expanse of the neuron related to gathering synaptic input from other neurons; these arborizations show some plasticity and can expand or regress, depending on the local neuronal microenvironment and the extent of input. The axon generally distributes to a relatively fixed set of specific target structures (neurons, muscle, and effector tissue) but also may expand or regress as dictated by demand. The internal state of gene expression is a snapshot of the specific demands to which the neuron is responding and may change according to hormonal or neurotransmitter signaling, or according to other molecular influences on transcription factors.

Neuronal Cell Types



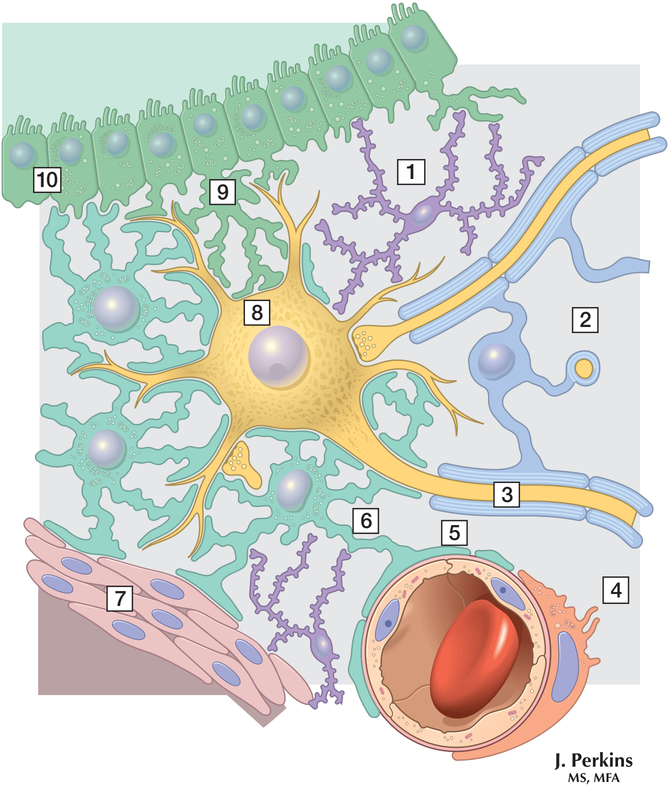
Red:	Motor neuron
Blue:	Sensory neuron
Purple:	Interneuron
Gray:	Glial and neurilemmal cells and myelin
CNS:	Central Nervous System
PNS:	Peripheral Nervous System

Note: Cerebellar cells not shown here

Neuronal Cell Types

1. Primary sensory unipolar ganglion cell of sensory cranial nerves
2. Primary sensory unipolar ganglion cell of dorsal root ganglion
3. Multipolar neuron (autonomic preganglionic neuron)
4. Autonomic ganglion cell
5. Schwann cell
6. Motor end plate (neuromuscular junction)
7. Myelin sheath
8. Myelinated motor axon
9. Multipolar neuron (spinal cord motor neuron)
10. Oligodendrocyte
11. Astrocyte
12. Multipolar neuron (pyramidal cell)

Comment: Neurons are organized into hierarchies. Incoming (afferent) information is transduced by sensory receptors associated with the distal portion of primary sensory neurons. These neurons convey the information into the central nervous system and synapse on secondary sensory neurons associated with reflex channels, cerebellar channels, and lemniscal channels, the latter of which is associated with conscious interpretation. Outputs from the central nervous system consist of (1) lower motor neurons, whose axons terminate on skeletal muscle fibers, forming neuromuscular junctions; and (2) preganglionic autonomic neurons of the sympathetic and parasympathetic nervous systems, whose axons synapse on ganglion cells that, in turn, regulate smooth muscle, cardiac muscle, secretory glands, metabolic cells, and cells of the immune system.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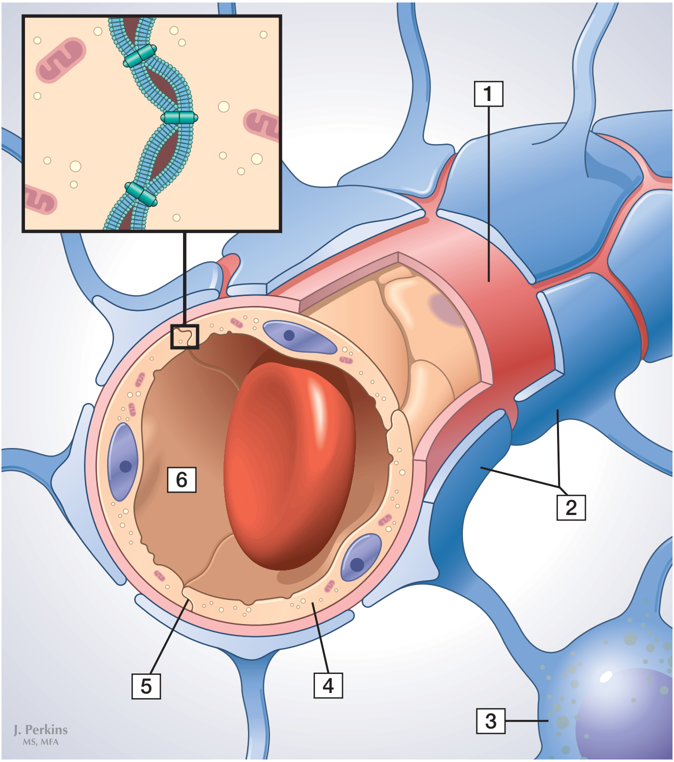
J. Perkins
MS, MFA

Glial Cell Types

1. Microglial cell
2. Oligodendrocyte
3. Axon
4. Perivascular pericyte
5. Astrocytic foot process
6. Astrocyte
7. Pia mater cells
8. Neuron
9. Tanycyte
10. Ependymal cell

Comment: Glial cells provide the supportive cellular structure and function for neurons. They act as scaffolding between neurons, sequester ions (potassium), insulate synaptic sites, provide trophic and molecular support for neurons (astrocytic functions), provide myelination of axons (oligodendrocyte function), phagocytize debris, participate in inflammatory responses, present antigens, and provide other immunologic and cytokine reactivity (microglial functions). Approximately 10 times more glial cells exist than neurons. Glial cells are the principal cell types that proliferate to form CNS tumors; neurons rarely form tumors.

Blood-Brain Barrier

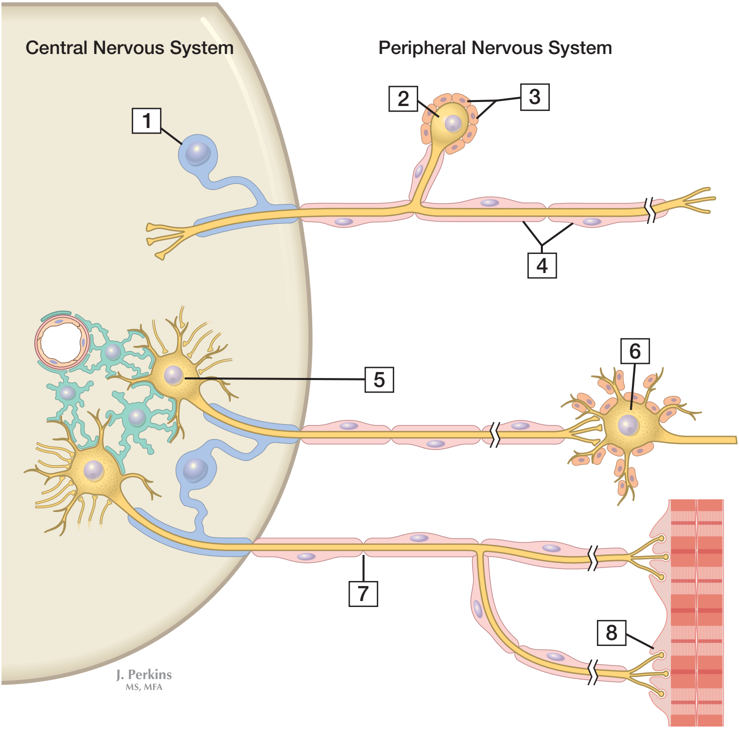


Blood-Brain Barrier

1. Basement membrane
2. Astrocytic foot process
3. Astrocyte
4. Capillary endothelial cell
5. Tight junction of endothelial cells
6. Capillary lumen

Comment: The major anatomical substrate for the blood-brain barrier (BBB) is the tight junctions of the capillary endothelial cells. They effectively keep large molecules out of the CNS and protect the brain from adverse effects of circulating toxins and potentially damaging molecules. Some substances can directly cross into the brain, others have a competitive facilitated transport (some amino acids), and others have an active transport system for ingress into the CNS. At sites of inflammation, tumors, trauma, and other insults to the brain, the BBB may be disrupted and allow damaging molecules into the brain. The effectiveness of the BBB prevents many therapeutic agents from having direct access to the brain and requires intraventricular or intrathecal delivery or coupling to a transport carrier.

Myelination of CNS and PNS Axons



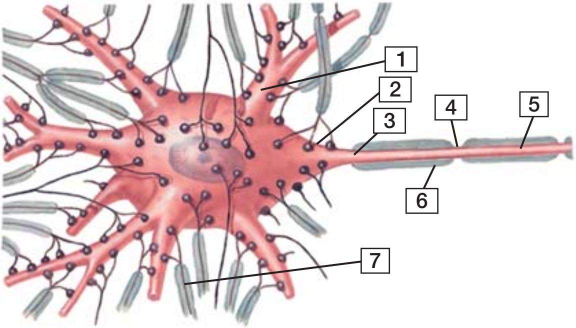
Myelination of CNS and PNS Axons

1. Oligodendrocyte
2. Primary sensory (ganglion) cell body
3. Satellite (supportive) cells
4. Schwann cells associated with myelin sheath
5. Preganglionic autonomic neuron
6. Postganglionic autonomic neuron
7. Nodes of Ranvier
8. Motor end plate (neuromuscular junction)

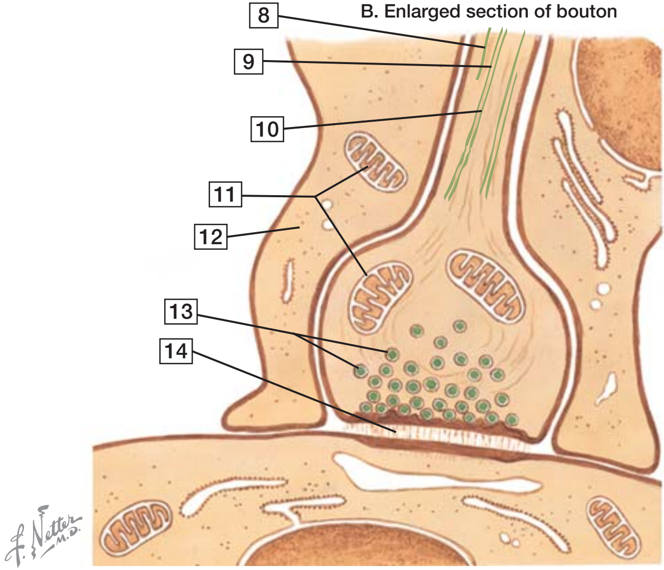
Comment: Peripheral axons greater than 1 to 2 μm diameter (primary sensory axons, motor axons, preganglionic autonomic axons) are myelinated by Schwann cells. A single Schwann cell enwraps a single internodal region of axon with a myelin sheath. Unmyelinated axons (small primary sensory axons, most postganglionic autonomic axons) are protected by a layer of Schwann cell cytoplasm. CNS axons are myelinated by oligodendrocytes; a single oligodendrocyte extends processes to multiple internodal regions and myelinates several such regions. Both Schwann cells and oligodendrocytes are capable of replicating in response to demyelinating events and can remyelinate axons, but the internodal distance of the new myelinated segment is usually shorter than the original myelinated segment.

Synaptic Morphology

A. Schematic of synaptic endings



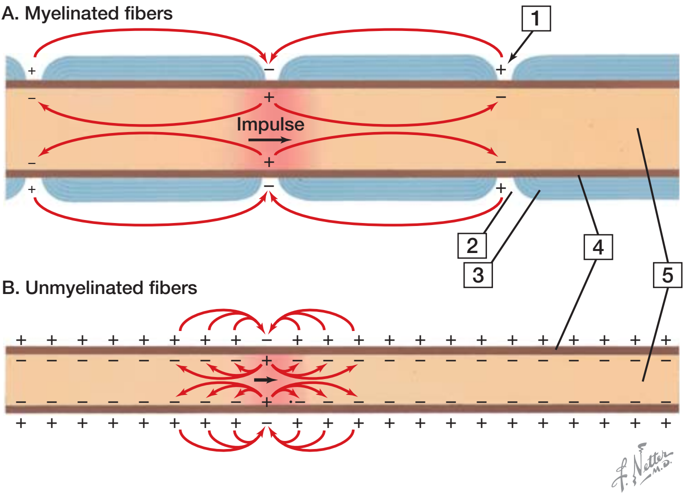
B. Enlarged section of bouton



Synaptic Morphology

1. Dendrite
2. Axon hillock
3. Initial segment
4. Nodes of Ranvier
5. Axon
6. Myelin sheath
7. Afferent axon ending on dendrite
8. Neurofilaments
9. Neurotubules
10. Axon (axoplasm)
11. Mitochondria
12. Glial process (astrocyte)
13. Synaptic vesicles
14. Synaptic cleft

Comment: The distribution of incoming synapses on a target neuron determines the source and excitability of that neuron. Some extensive synapses (serial) or synapses near the initial segment of the axons exert a disproportional influence on the eventual firing of action potentials by the target neuron. In general, synaptic vesicles fuse with the axonal membrane at the synaptic cleft upon neuronal depolarization and the accompanying influx of calcium, triggering the simultaneous release of many quanta (vesicular content) of neurotransmitter. An axon terminal may release more than one neurotransmitter, depending upon the frequency of axonal firing and the local and distant influences (e.g., induction) on gene expression in the cell of origin. Neurotransmitter release is not necessarily linear with frequency of firing. Also, in the CNS, there is often a mismatch between sites at which neurotransmitter is released and the presence of cognate receptors for that neurotransmitter on potential target neurons. These factors collectively contribute to the complexity of synaptic signaling in the brain.


A. Myelinated fibers
1
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Impulse
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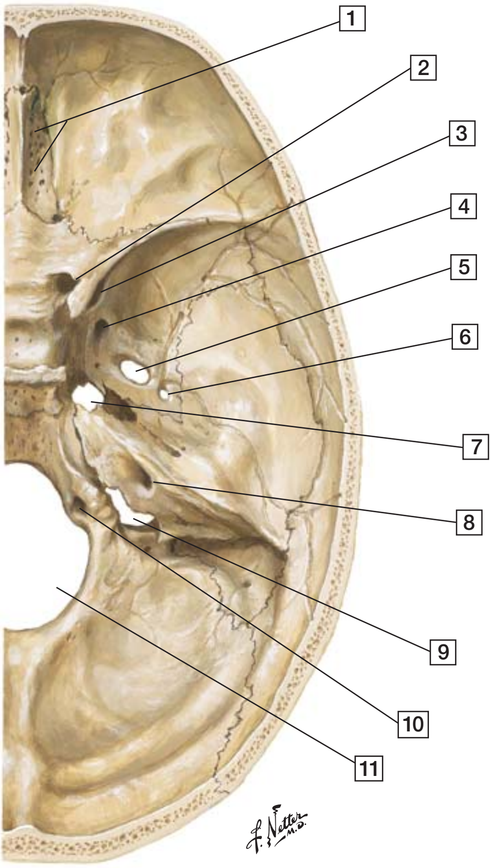
B. Unmyelinated fibers
f. Netter M.D.

Conduction Velocity

1. Node (site of action potential reinitiation)
2. Nodes of Ranvier
3. Myelin sheath
4. Axolemma
5. Axoplasm

Comment: An action potential is triggered in a neuronal cell membrane when sufficient depolarization has occurred to allow the influx of sodium ions to overwhelm the capacity of the efflux of potassium ions to counter the influx of positive charge. This results in an explosive depolarization that moves the neuronal cell membrane toward its sodium equilibrium potential (approximately +55 mV). An action potential at one node exerts enough local influence on the adjacent node (and perhaps several distant nodes) to bring those sites to a depolarized state sufficient to trigger an action potential. This process reinitiates the action potential at each node. If a local anesthetic is used to block the excitability (capacity to depolarize) at several successive nodes, then a conduction block will occur and propagation of the action potential will cease. The conduction velocity of an axon depends upon the axonal diameter, also a reflection of the extent of myelination.

Foramina in the Base of the Adult Skull

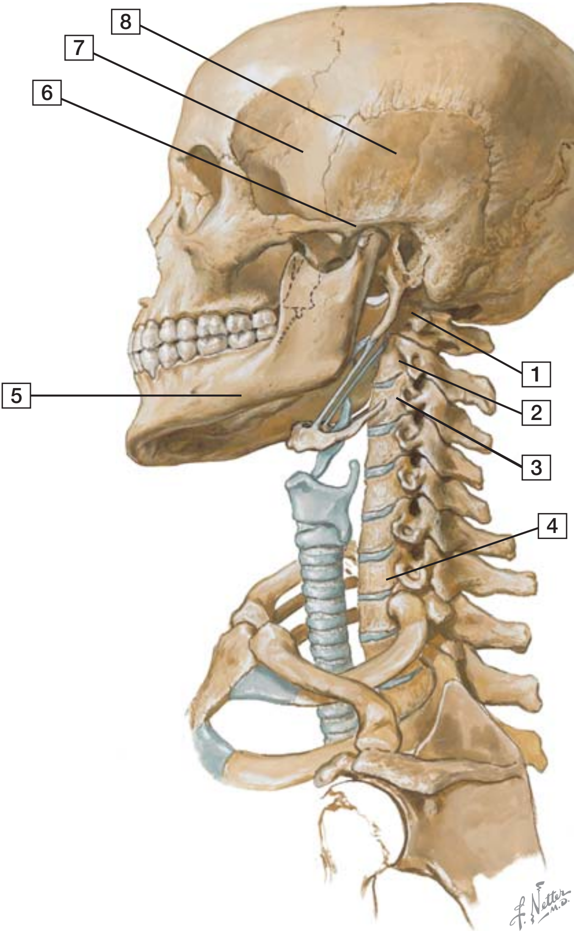


Foramina in the Base of the Adult Skull

Each number denotes the foramen and major structures that pass through it.

1. Foramina of cribriform plate (olfactory nerve bundles)
2. Optic canal (optic nerve, ophthalmic artery)
3. Superior orbital fissure (CNs III, IV, VI), ophthalmic nerve (V1), superior ophthalmic vein
4. Foramen rotundum (maxillary nerve [V2])
5. Foramen ovale (mandibular nerve [V3], accessory meningeal artery, lesser petrosal artery (occasionally)
6. Foramen spinosum (middle meningeal artery and vein, meningeal branch of mandibular nerve)
7. Foramen lacerum (internal carotid artery, internal carotid nerve plexus)
8. Internal acoustic meatus (CNs VII and VIII, labyrinthine artery)
9. Jugular foramen (CNs IX, X, XI; inferior petrosal sinus, sigmoid sinus, posterior meningeal artery)
10. Hypoglossal canal (CN XII)
11. Foramen magnum (medulla, meninges, vertebral arteries, spinal roots of CN XI)

Bony Framework of the Head and Neck

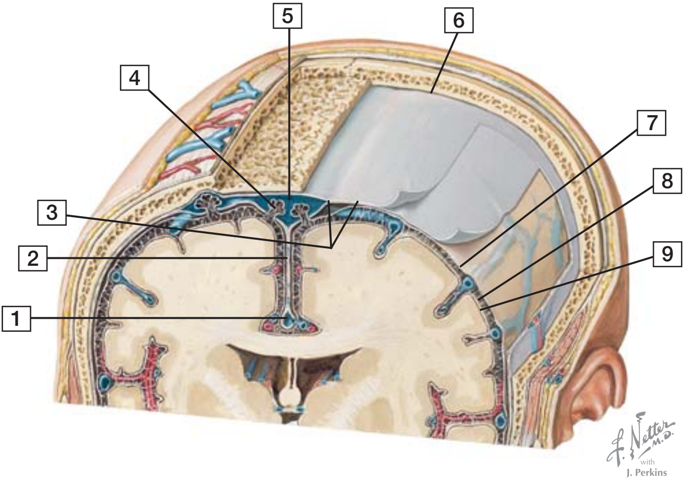


Bony Framework of the Head and Neck

1. Atlas (C1)
2. Axis (C2)
3. C3 vertebra
4. C7 vertebra
5. Mandible
6. Zygomatic arch
7. Sphenoid bone
8. Temporal bone

Comment: The cervical vertebrae are well articulated with each other and provide a strong bony framework for protecting the spinal cord as it emerges from the brain stem. But the sites of articulation and the vertebrae themselves are sites of potential vulnerability to trauma and can be fractured or dislocated, causing spinal cord injury. Cervical discs may herniate, resulting in protrusion of the nucleus pulposus and possible impingement on sensory roots (radiating pain) or motor roots (motor weakness) that emerge through the intervertebral foramina.

Schematic of the Meninges and Their Relationships to the Brain and Skull

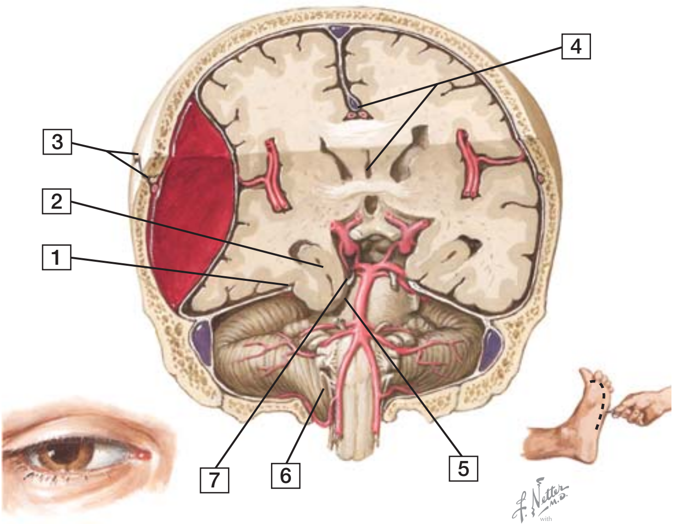


Schematic of the Meninges and Their Relationships to the Brain and Skull

1. Inferior sagittal sinus
2. Falx cerebri
3. Inner and outer layers of dura mater
4. Arachnoid granulations
5. Superior sagittal sinus
6. Epidural space (a potential space)
7. Arachnoid membrane
8. Subarachnoid space
9. Pia mater

Comment: The pia mater adheres to every contour of the brain, while the delicate arachnoid membrane spans these contours. The resultant subarachnoid space is a site in which cerebrospinal fluid (CSF) is found, providing buoyancy and protection to the brain, and also a site in which the cerebral arteries and veins travel. A ruptured berry aneurysm results in an arterial bleed into the subarachnoid space. The dura mater is a tough protective membrane that adheres below to the arachnoid, and above to the skull, with a split into an inner and outer layer for the venous sinuses. These sinuses contain the draining venous blood, which is channeled into the additional sinuses and subsequently into the internal jugular vein. Arachnoid granulations protrude through the inner layer of the dura to drain CSF into the venous sinuses via one-way valves. Trauma may tear the bridging veins that drain into the sinuses, resulting in a subdural hematoma (subdural space is a potential space). A skull fracture that tears a meningeal artery may result in a bleed into the epidural space, a potential space between the outer layer of the dura and the skull.

Hematomas

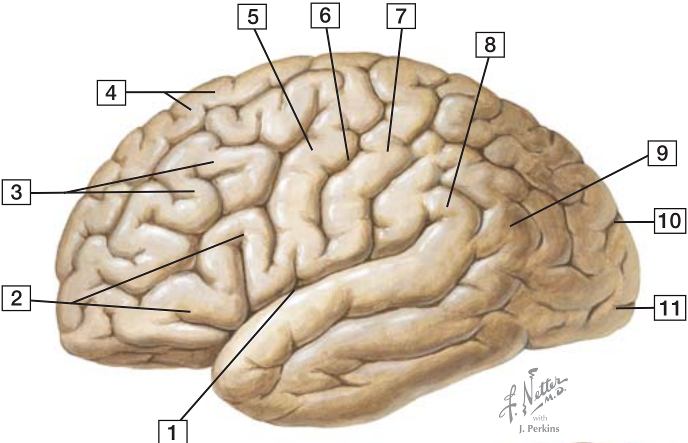


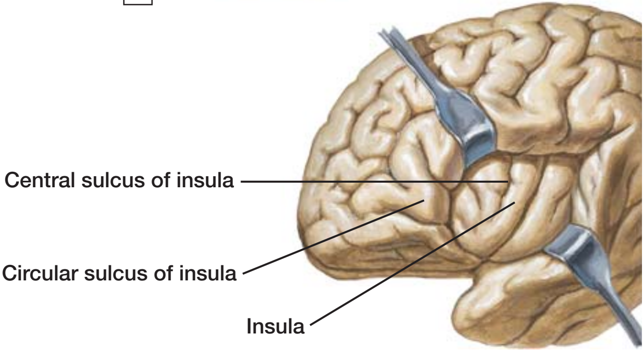
Hematomas

1. Tentorium cerebelli
2. Herniation of temporal lobe
3. Skull fracture affecting middle meningeal artery
4. Midline shift of falx cerebri and third ventricle
5. Compression of cerebral peduncle
6. Herniation of cerebellar tonsil in foramen magnum
7. Compression of CN III

Comment: A skull fracture that tears the middle meningeal artery can result in an arterial bleed into the epidural space. This bleed, or any space-occupying mass in the supratentorial space, can displace the brain and cause it to shift midline structures (subfalcial) or compress downward, herniating the temporal lobe across the tentorium cerebelli (transtentorial herniation). The herniating brain often compresses CN III, causing an ipsilateral fixed, dilated pupil and oculomotor palsy, and it compresses the adjacent cerebral peduncle, causing contralateral hemiparesis and contralateral drooping lower face (central facial palsy). A space-occupying mass in the posterior fossa can herniate the cerebellar tonsil through the foramen, compressing the brain stem, altering vital visceral functions such as blood pressure and respiration, and can result in death.

Surface Anatomy of the Forebrain: Lateral View





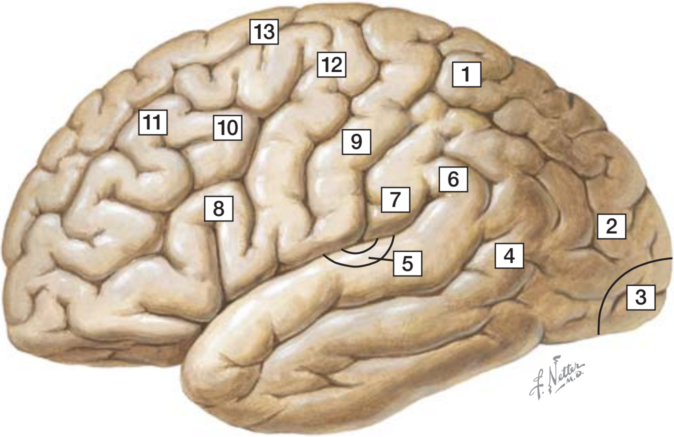
Surface Anatomy of the Forebrain: Lateral View

1. Lateral (Sylvian) fissure
2. Inferior frontal gyrus
3. Middle frontal gyrus
4. Superior frontal gyrus
5. Precentral gyrus
6. Central sulcus
7. Postcentral gyrus
8. Supramarginal gyrus
9. Angular gyrus
10. Parietooccipital sulcus
11. Calcarine fissure

Comment: Some specific functional capabilities are localized to particular gyri or regions of the cerebral cortex, such as somatic motor control (precentral gyrus); somatosensory and trigeminal sensory processing (postcentral gyrus); auditory processing (transverse gyrus of Heschl); visual processing (occipital cortex on the banks of the calcarine fissure); expressive language function (left inferior frontal gyrus, Broca's area); and receptive language function (supramarginal gyrus and adjacent areas, Wernicke's area). Other important functional capabilities are more diffuse, such as long-term memory storage and complex intellectual capabilities.

See book 3.1, upper figure and lower right figure

Lateral View of the Forebrain: Functional Regions

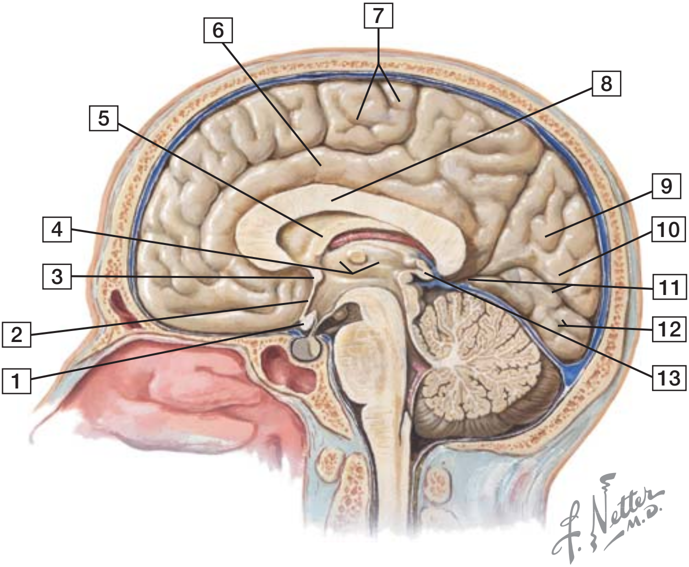


Lateral View of the Forebrain: Functional Regions

1. Superior parietal lobule
2. Visual association cortex
3. Primary visual cortex
4. Multisensory association areas of cortex
5. Primary auditory cortex
6. Wernicke's area
7. Secondary somatosensory cortex
8. Broca's area
9. Primary somatosensory cortex
10. Premotor cortex
11. Frontal eye fields
12. Primary motor cortex
13. Supplemental motor cortex

Comment: The primary motor cortex and primary sensory cortex demonstrate contralateral topographic localization of specific regions of the body. The lower extremities are represented medially in the paracentral lobule, and the body is represented in slightly more lateral regions. The upper extremity is represented laterally, and the face is represented far laterally, just above the lateral fissure. The perioral region and the fingers and hands have a disproportionally large volume of cortex processing information, perhaps reflecting the survival value and functional importance of information from these regions, and the ability to control important related musculature. Even into adulthood, cortical plasticity can occur. The amputation or loss of a specific region, such as a digit, results in the former processing regions for that information demonstrating plasticity by taking over processing of information from adjacent topographically regions close to the site of loss.

Anatomy of the Medial (Midsagittal) Surface of the Brain In Situ

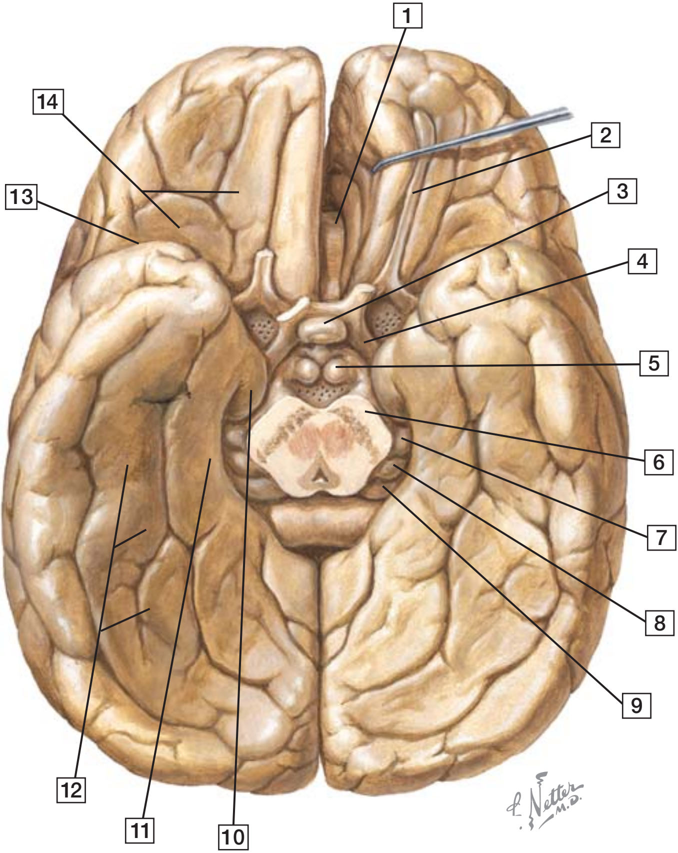


Anatomy of the Medial (Midsagittal) Surface of the Brain In Situ

1. Optic chiasm
2. Lamina terminalis
3. Anterior commissure
4. Thalamus
5. Fornix
6. Cingulate gyrus
7. Paracentral lobule
8. Corpus callosum
9. Cuneus
10. Calcarine cortex, upper bank
11. Calcarine sulcus (fissure)
12. Calcarine cortex, lower bank
13. Pineal gland

Comment: On a midsagittal view of the brain, the commissural bundles are visible (corpus callosum, anterior commissure, posterior commissure); all of the structures of the brain stem are visible (medulla, pons, midbrain), as well as the thalamus and hypothalamus; and the components of the ventricular system are visible (third ventricle and the interventricular foramen of Monro opening into the more laterally located lateral ventricles, the aqueduct, the fourth ventricle, the caudal openings into the subarachnoid space [foramina of Magendie and of Luschka] and some of the subarachnoid cisterns [cisterna magna]).

Anatomy of the Basal Surface of the Brain, with the Brain Stem and Cerebellum Removed



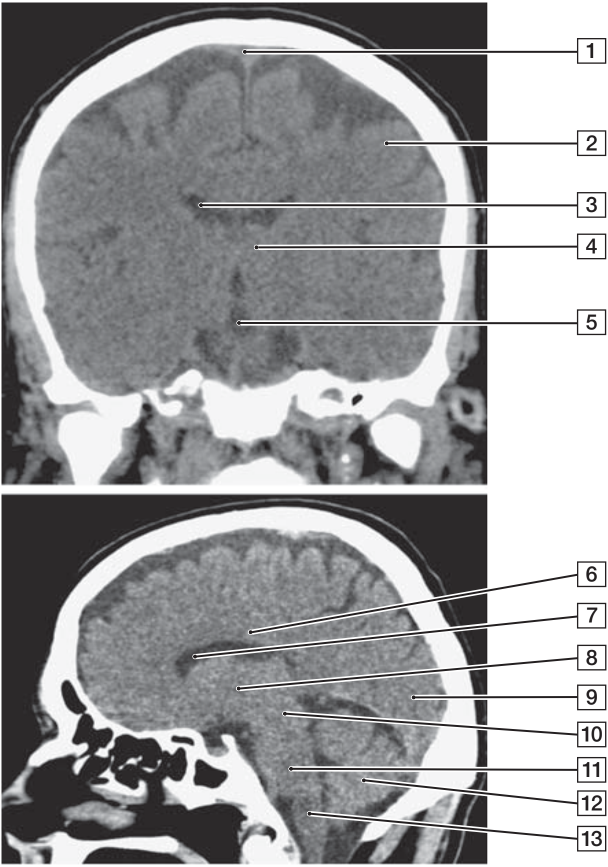
Anatomy of the Basal Surface of the Brain, with the Brain Stem and Cerebellum Removed

1. Genu of corpus callosum
2. Olfactory tract
3. Pituitary gland
4. Optic tract
5. Mammillary bodies
6. Cerebral peduncles (crus cerebri)
7. Lateral geniculate body (nucleus)
8. Medial geniculate body (nucleus)
9. Pulvinar
10. Uncus
11. Parahippocampal gyrus
12. Medial occipitotemporal gyrus
13. Lateral (Sylvian) fissure
14. Orbital gyri

Comment: A basal view of the cerebral cortex reveals some limbic structures, such as the olfactory bulb and tract, mammillary bodies, uncus, and parahippocampal gyrus, as well as temporal lobe cortical regions under which the amygdala (rostrally) and the hippocampal formation (middle region) are found. Several important thalamic regions are also directly visible, such as the medial geniculate body (auditory processing), lateral geniculate body (visual processing), and the pulvinar (some visual processing).

Brain Imaging: Computed Tomography Scans, Coronal and Sagittal

A. Coronal view



B. Sagittal view

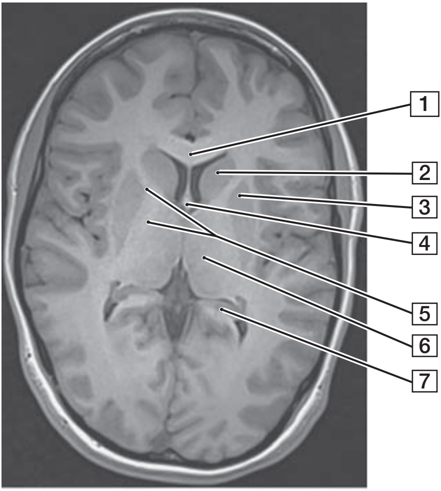
Brain Imaging: Computed Tomography Scans, Coronal and Sagittal

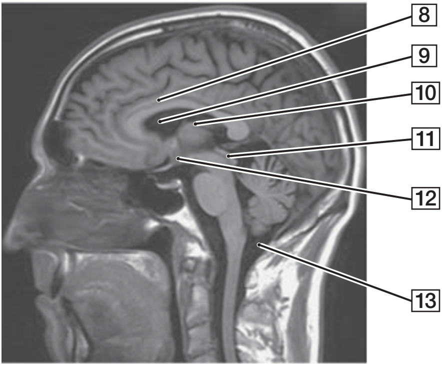
1. Superior sagittal sinus
2. Cortical gyrus
3. Lateral ventricle
4. Thalamus
5. Third ventricle
6. Corpus callosum
7. Lateral ventricle
8. Thalamus
9. Occipital lobe
10. Midbrain
11. Pons
12. Cerebellum
13. Medulla

Comment: Computed tomography (CT) scan is an x-ray–based imaging technique that allows visualization of slices through the brain in any plane at desired thicknesses. It is particularly useful for delineating differences in tissue density and showing soft tissue, fluid, and bone. CT imaging can be used with contrast to image blood vessels or to reveal the presence of a tumor on the basis of the disruption of the blood-brain barrier.

Brain Imaging: Magnetic Resonance Imaging, Axial and Sagittal T1-Weighted Images

A. Axial view





B. Sagittal view

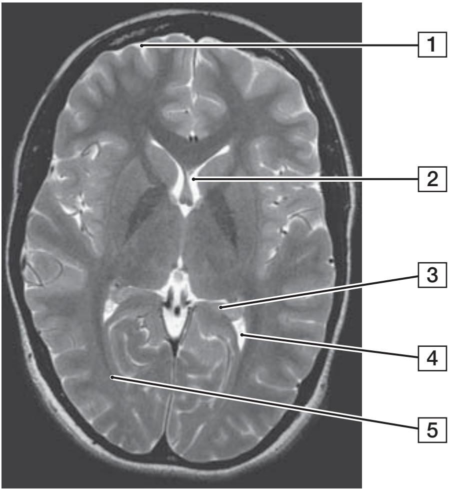
Brain Imaging: Magnetic Resonance Imaging, Axial and Sagittal T1-Weighted Images

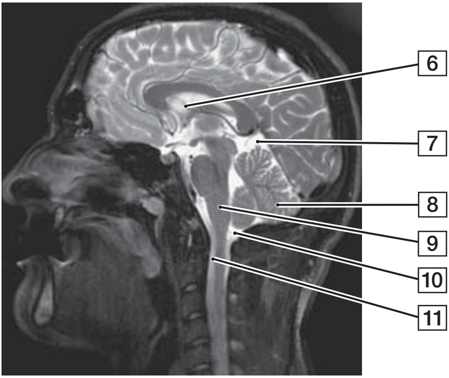
1. Genu, corpus callosum
2. Head of caudate nucleus
3. Putamen
4. Columns of the fornix
5. Internal capsule
6. Thalamus
7. Hippocampal formation
8. Cingulate cortex
9. Lateral ventricle
10. Fornix
11. Colliculi
12. Hypothalamus
13. Cisterna magna

Comment: Magnetic resonance (MR) imaging uses short bursts (radiofrequency pulses) of electromagnetic waves that are absorbed by protons in the tissues of the patient in the scanner. The pulses cause alignment of the protons as a result of raised energy levels. During the relaxation phase, a detector records the emitted energy and a uniform image of the tissue is obtained. Short repetition times and echo times are termed T1-weighted (ventricles appear dark), and longer repetition times and echo times are termed T2-weighted (ventricles appear white). High-resolution T1-weighted MR images reveal detailed internal anatomy and are used throughout *Netter's Atlas of Neuroscience* (2nd ed.) to show cross-sectional brain stem anatomy (on line), axial anatomy, coronal anatomy, and sagittal anatomy (on line).

Brain Imaging: Magnetic Resonance Imaging, Axial and Sagittal T2-Weighted Images

A. Axial view





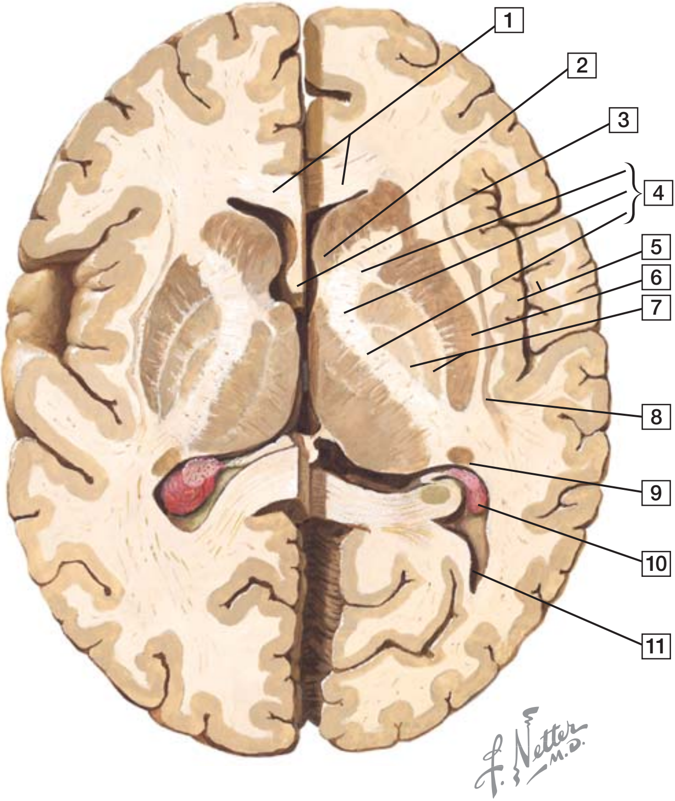
B. Sagittal view

Brain Imaging: Magnetic Resonance Imaging, Axial and Sagittal T2-Weighted Images

1. Subarachnoid space
2. Frontal pole, lateral ventricles
3. Hippocampal formation
4. Temporal pole, lateral ventricle
5. Optic radiations
6. Site of lateral ventricle
7. Subarachnoid space
8. Cerebellum
9. Medulla
10. Cisterna magna
11. Subarachnoid space

Comment: The T2-weighted magnetic resonance image shows the ventricular system and cerebrospinal fluid in the subarachnoid space (cisterns) as white. These images are useful for revealing pathology such as infarcts, tumors, edema, and demyelination. A contrast agent such as gadolinium can be used to delineate a tumor because of its ability to leak across a disrupted blood-brain barrier.

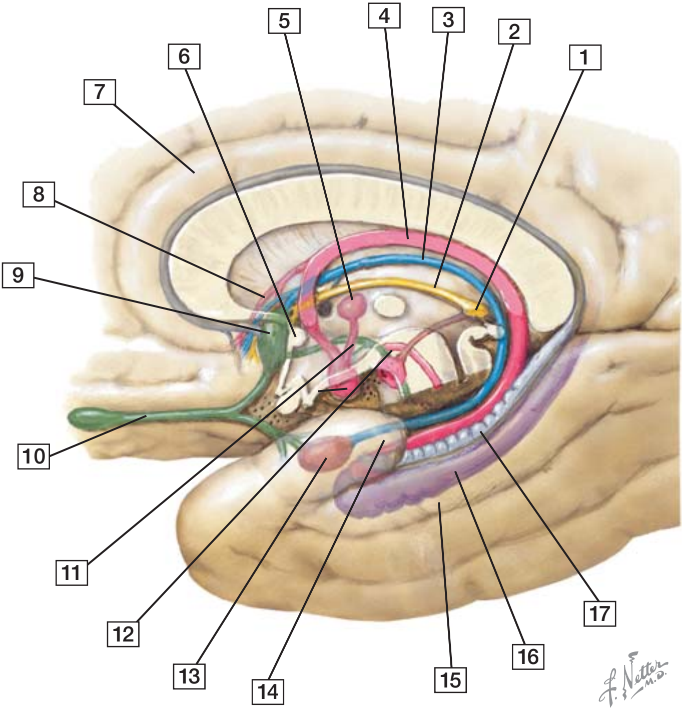
Horizontal Brain Sections Showing the Basal Ganglia



Horizontal Brain Sections Showing the Basal Ganglia

1. Genu of corpus callosum
2. Head of caudate nucleus
3. Columns of the fornix
4. Internal capsule (anterior limb, genu, posterior limb)
5. Insular cortex
6. Putamen
7. Globus pallidus
8. Claustrum
9. Tail of caudate nucleus
10. Choroid plexus of lateral ventricle
11. Occipital (posterior) horn of lateral ventricle

Comment: The basal ganglia are a collection of telencephalic nuclei (caudate nucleus, putamen, globus pallidus) and their associated tracts, as well as selected associated subtelencephalic nuclei (subthalamus, substantia nigra). Collectively, the basal ganglia share loops of circuitry with the thalamus and associated cortical regions and assist the cortex in selecting wanted subroutines of activity and suppressing unwanted subroutines of activity. The basal ganglia are particularly conspicuous when damaged, related to movement disorders (tremors, rigidity, choreoathetosis, dystonia, ballismus), although the basal ganglia are involved in modulating activity in many types of cortical function other than just motor function. The axial (horizontal) sections are particularly useful for revealing the association of basal ganglia telencephalic nuclei with the thalamus and intervening internal capsule.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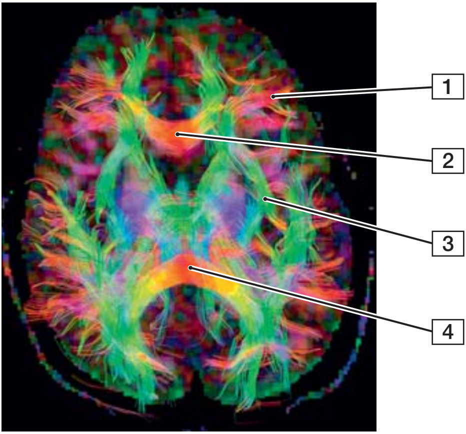
Major Limbic Forebrain Structures

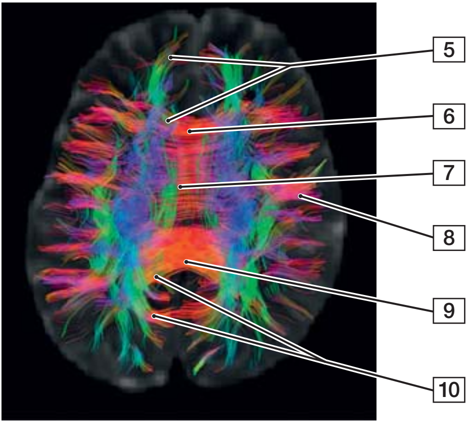
1. Habenula
2. Stria medullaris
3. Stria terminalis
4. Fornix
5. Anterior nucleus of the thalamus
6. Anterior commissure
7. Cingulate cortex
8. Precommissural fornix
9. Septal nuclei
10. Olfactory tract
11. Mammillothalamic tract
12. Median forebrain bundle
13. Amygdala (nuclei)
14. Uncus
15. Parahippocampal gyrus
16. Hippocampus
17. Dentate gyrus

Comment: The limbic system was named for a ring (limbus) of structures that encircle the thalamus and associate cortical, as well as subcortical, circuitry in functionally subserving tasks related to emotional responsiveness and behavior, motivational and reward behavior, consolidation of short-term memory, and environmentally appropriate and situationally appropriate regulation of visceral (hypothalamic and autonomic) and hormonal control of appetitive and autonomic functions. The limbic system provides a contextually appropriate individualized response to stimuli and challenges in the environment. Some limbic components form C-shaped pathways from the temporal lobe into more rostral structures of the diencephalon and telencephalon (hippocampal formation and associated fornix; amygdala and associated stria terminalis).

Color Imaging of the Corpus Callosum by Diffusion Tensor Imaging

Axial view





Axial view

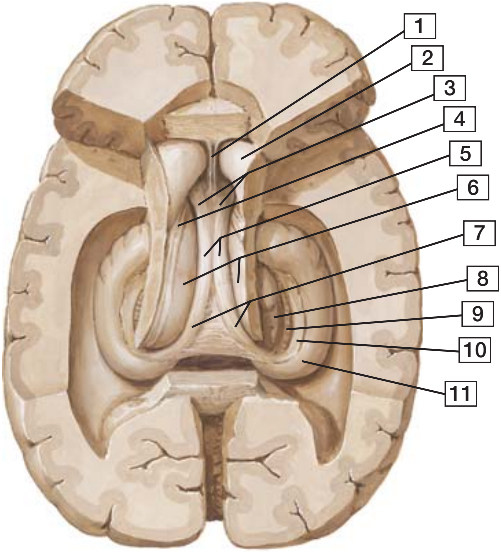
Color Imaging of the Corpus Callosum by Diffusion Tensor Imaging

1. Lateral fibers of the corpus callosum
2. Genu of the corpus callosum
3. Cortical association fibers
4. Splenium of the corpus callosum
5. Forceps minor
6. Genu of the corpus callosum
7. Body of the corpus callosum
8. Lateral fibers of the corpus callosum
9. Splenium of the corpus callosum
10. Forceps major

Comment: The corpus callosum connects similar regions of the two cerebral hemispheres with each other. These connections permit each hemisphere to be aware of stimuli or activities related to both sides of the body. Without such connections, a visual stimulus from the left visual field, interpreted by the right hemisphere, would not be noted by the left hemisphere; the left hemisphere would be unable to verbally describe the stimulus, nor would the left hemisphere be able to direct the right side of the body to react to the stimulus.
On occasion, significant damage to the corpus callosum occurs, or congenital agenesis occurs, separating the two hemispheres (split brain). The corpus callosum (and other commissures) are sometimes surgically severed for seizure disorders to prevent the spread of the seizure to the mirror image region of the opposite hemisphere.

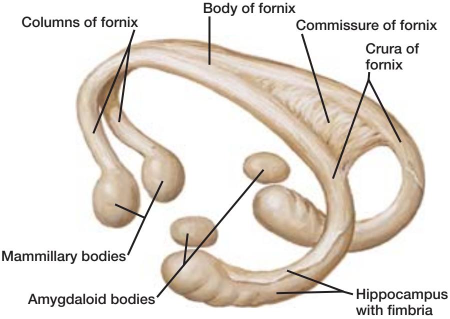
Hippocampal Formation and Fornix

A. Dissection of the hippocampal formation and fornix



F. Netter M.D. with J. Perkins MS, MFA

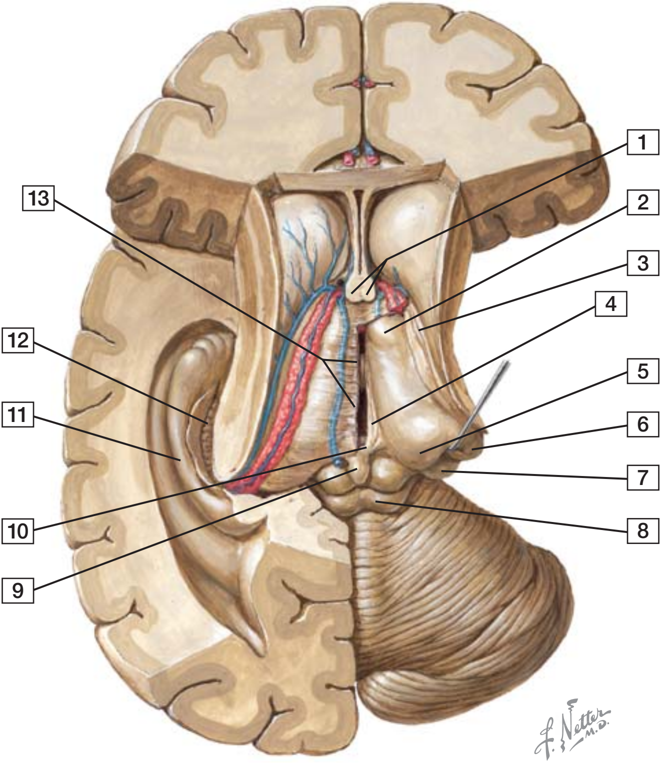
B. 3-D reconstruction of the fornix



Hippocampal Formation and Fornix

1. **1.** Septum pellucidum
2. **2.** Head of caudate nucleus
3. **3.** Columns of fornix
4. **4.** Stria terminalis
5. **5.** Body of fornix
6. **6.** Thalamus
7. **7.** Crura of fornix
8. **8.** Parahippocampal gyrus
9. **9.** Dentate gyrus
10. **10.** Fimbria of hippocampus
11. **11.** Hippocampus

Comment: The hippocampal formation, with its connections to the diencephalon and telencephalon through the fornix, is a C-shaped structure that anatomically sweeps from the temporal lobe around the thalamus into rostral regions of the forebrain. The hippocampal formation is necessary for the consolidation of short-term memory into permanent or long-term traces. Information from the hippocampal formations proceeds from the pyramidal neurons of the entorhinal cortex to granule cells in the dentate gyrus, to CA3 pyramidal neurons, to CA1 and CA2 pyramidal neurons, to the subiculum, and back to the entorhinal cortex. CA neurons (especially CA1) are particularly vulnerable to ischemia; a disruption in any part of the circuitry can disrupt the functional integrity of the hippocampal formation.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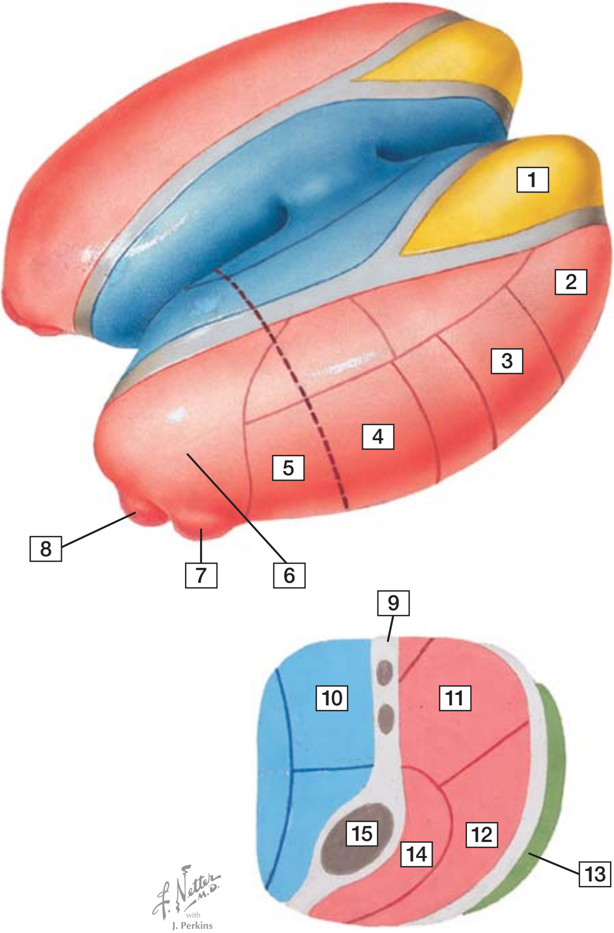
Thalamic Anatomy

1. Columns of fornix
2. Anterior tubercle
3. Stria terminalis
4. Stria medullaris
5. Pulvinar
6. Lateral geniculate body
7. Medial geniculate body
8. Inferior colliculus
9. Pineal
10. Habenular commissure
11. Hippocampus
12. Dentate gyrus
13. Third ventricle

Comment: The thalamus is reciprocally connected with the cerebral cortex; some nuclei (specific) interconnect with specific and circumscribed regions of the cortex, and other nuclei (nonspecific) have diffuse connections throughout the cortex. These nonspecific regions, through connections with the brain stem reticular formation, alert the cerebral cortex and provide the physiological substrate for consciousness. Tracts and nuclei of the limbic system and basal ganglia surround the thalamus and interact with thalamo-cortical circuitry.

Thalamic Nuclei

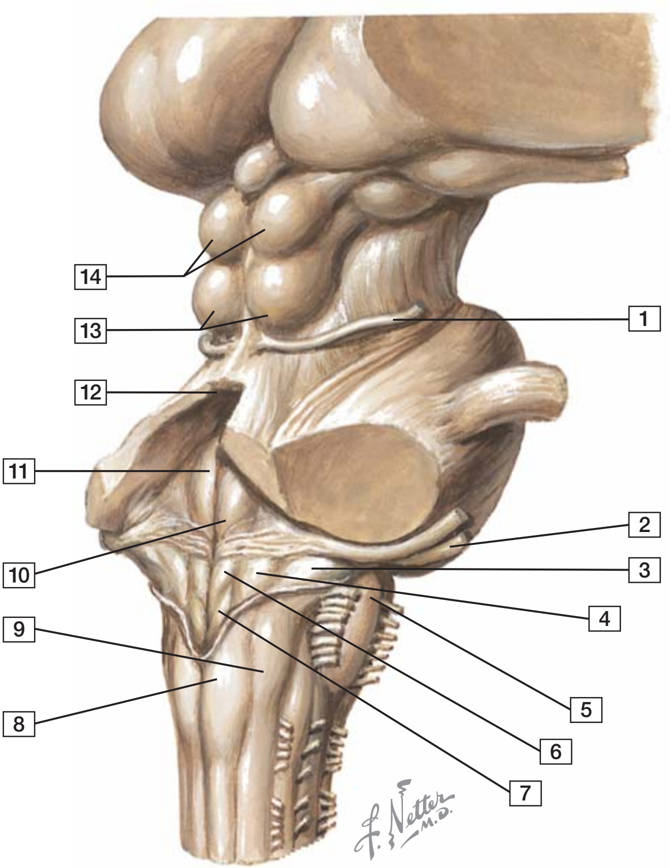


Thalamic Nuclei

1. Anterior nuclei
2. Ventral anterior (VA) nucleus
3. Ventrolateral (VL) nucleus
4. Ventral posteromedial (VPM) nucleus
5. Ventral posterolateral (VPL) nucleus
6. Pulvinar
7. Lateral geniculate body (nucleus)
8. Medial geniculate body (nucleus)
9. Internal medullary lamina
10. Medial dorsal nucleus (MD)
11. Lateral posterior (LP) nucleus
12. Ventral posterolateral (VPL) nucleus
13. Reticular nucleus
14. Ventral posteromedial (VPM) nucleus
15. Centromedian (CM) nucleus

Comment: Many of the specific thalamic nuclei receive topographically organized information from their sources of input (e.g., sensory lemniscal channels for specific sensory nuclei for somatosensory, trigeminal sensory, auditory, and visual processing). The thalamus is not merely a relay station to the cortex; it extracts information from its input and sends it to the associated region of cerebral cortex in discrete patterns of projections. The cortex, in turn, can act through interneurons of the thalamus to fine-tune its own input that it is receiving from the thalamus. With many thalamic nuclei, damage to the nucleus itself produces symptoms similar to damage to its associated cortical region. This is also the case for nuclei such as MD, interconnected with regions of frontal cortex.

Brain Stem Surface Anatomy: Posterolateral View

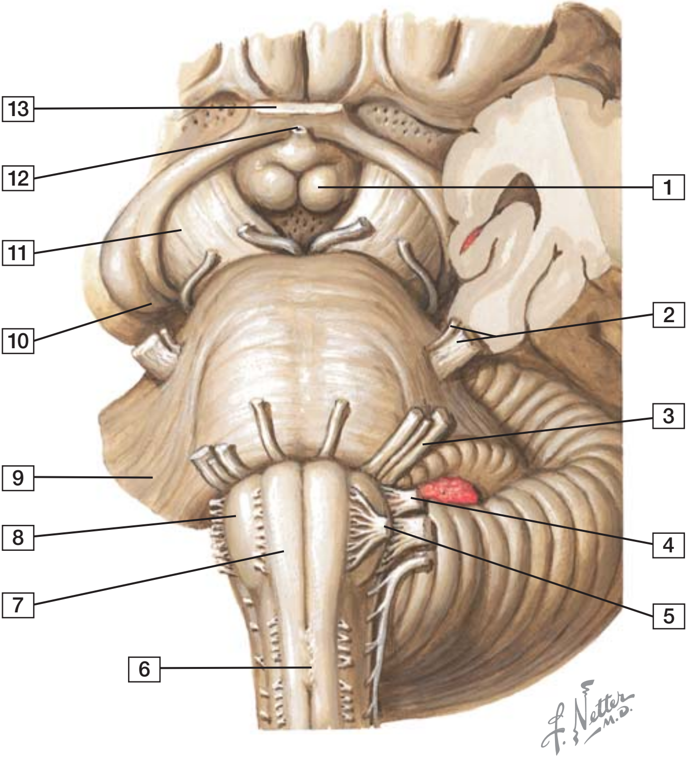


Brain Stem Surface Anatomy: Posterolateral View

1. Trochlear nerve (CN IV)
2. Facial nerve (CN VII)
3. Inferior cerebellar peduncle
4. Vestibular area
5. Olive
6. Hypoglossal trigone
7. Vagal trigone
8. Gracile tubercle
9. Cuneate tubercle
10. Facial colliculus
11. Medial eminence
12. Superior medullary velum
13. Inferior colliculus
14. Superior colliculus

Comment: Eminences, tubercles, trigones, and regions of brain stem on the floor of the fourth ventricle or regions adjacent to the fourth ventricle denote the presence of important underlying nuclei and associated pathways (gracile and cuneate tubercles—nuclei gracilis and cuneatus; vestibular area—vestibular nuclei; hypoglossal and vagal trigones—CN nuclei XII and X; facial colliculus—CN nucleus VI [abducens] and dorsally looping axons of CN VII arising from CN nucleus VII). Involvement of these brain stem regions from tumors, infarcts, or other damage is reflected by specific deficits related to the functions of the underlying sensory, motor, or autonomic nuclei and pathways.

Brain Stem Surface Anatomy: Anterior View

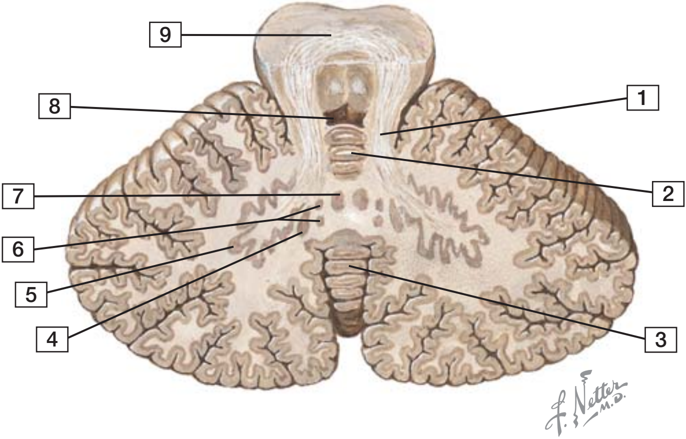


Brain Stem Surface Anatomy: Anterior View

1. Mammillary bodies
2. Trigeminal nerve (CN V)
3. Vestibulocochlear nerve (CN VIII)
4. Glossopharyngeal nerve (CN IX)
5. Hypoglossal nerve (CN XII)
6. Pyramidal decussation
7. Pyramid
8. Olive
9. Middle cerebellar peduncle
10. Lateral geniculate body
11. Cerebral peduncle
12. Infundibular stalk
13. Optic chiasm

Comment: All of the peripheral cranial nerves (III to XII) emerge from the ventral (anterior) surface of the brain stem except CN IV, which emerges from the dorsal surface of the midbrain. These cranial nerves are subject to damage or insults both peripherally (e.g., acoustic schwannoma impinging on CNs VII and VIII) and centrally (e.g., brain stem infarct in a paramedian artery damaging CN axons of XII or VI in their central location within the brain stem). The medullary pyramids are a conspicuous landmark denoting the presence of the corticospinal tract and its decussation at the medullary–spinal cord boundary. Damage to the medullary pyramid in isolation is unusual and is accompanied by loss of skilled hand movements contralaterally and mild decrease in tone. This is in contrast to damage to the corticospinal tract in the internal capsule, cerebral peduncle, or lateral funiculus of the spinal cord, where damage involves other descending pathways with motor consequences, resulting in spastic paresis, increased tone, and increased reflexes contralaterally (internal capsule, cerebral peduncles) or ipsilaterally (lateral funiculus of the spinal cord).

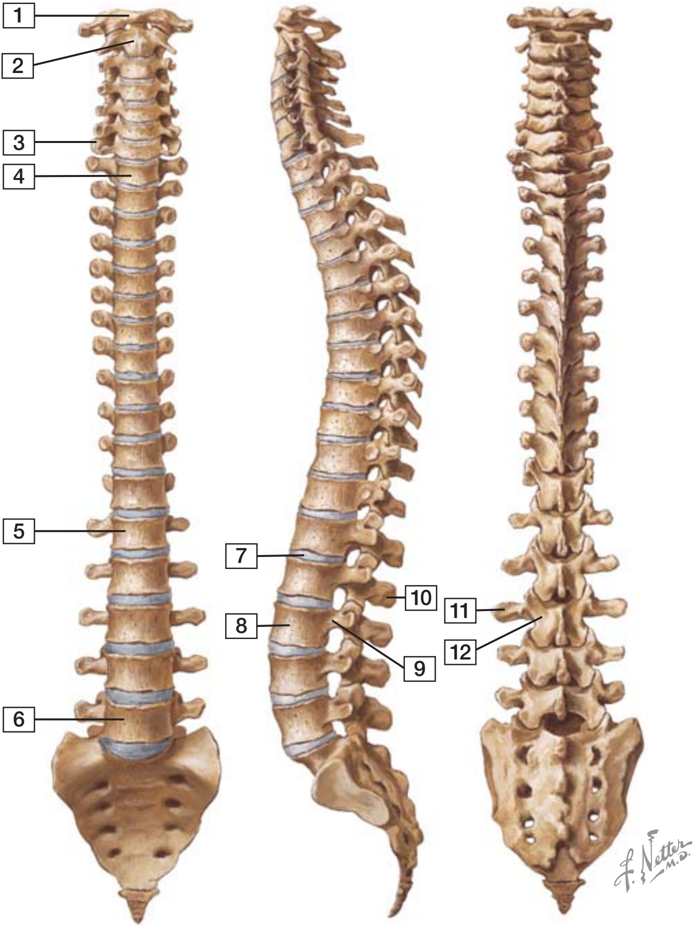
Cerebellar Anatomy: Internal Features



Cerebellar Anatomy: Internal Features

1. Superior cerebellar peduncle
2. Lingula
3. Vermis
4. Emboliform nucleus
5. Dentate nucleus
6. Globose nucleus
7. Fastigial nucleus
8. Fourth ventricle
9. Decussation of the superior cerebellar peduncles

Comment: The cerebellum helps to smooth and coordinate movements as its most conspicuous function, and it also coordinates neural activities associated with nonmotor functions. The deep cerebellar nuclei provide the coarse adjustment, and the cerebellar cortex provides the fine adjustment through processing of inputs and subsequent modulation of the deep nuclei. The deep nuclei help to smooth and coordinate motor functions through projections to upper motor neuronal structures [fastigial nucleus with vestibulospinal systems; globose and emboliform nuclei with the rubrospinal tract; dentate nucleus with the corticospinal tract mainly via (ventrolateral nucleus) of the thalamus]. Lesions of the deep nuclei are more severe and lasting in producing loss of coordination, dysmetria, ataxia, and other associated cerebellar symptoms than are lesions of regions of the cerebellar cortex.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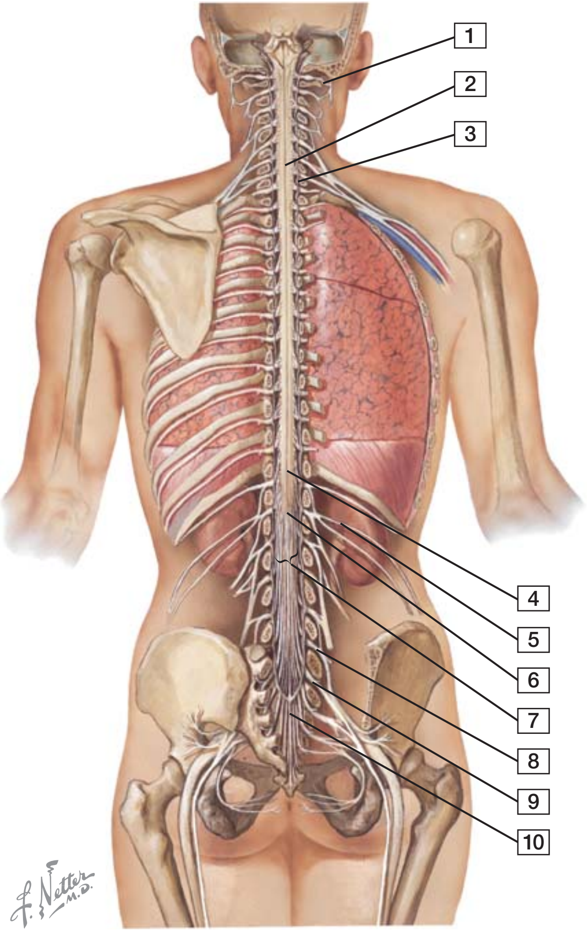
Spinal Column: Bony Anatomy

1. Atlas (C1 vertebra)
2. Axis (C2 vertebra)
3. C7 vertebra
4. T1 vertebra
5. L1 vertebra
6. L5 vertebra
7. Intervertebral disc
8. Vertebral body
9. Pedicle
10. Spinous process
11. Transverse process
12. Lamina

Comment: The vertebral bodies articulate with each other and are cushioned by intervertebral discs. These discs protect vertebrae and act as shock absorbers, and provide for flexibility of the spine. If the centrally located nucleus pulposus of the intervertebral discs protrudes through the tough protective annulus (herniated disc), it can impinge on emerging nerve roots (especially a dorsal root at the foramen, causing shooting, excruciating pain in a dermatomal pattern and also possible motor damage if it impinges on a ventral root). A herniated cervical disc also can impinge on the adjacent spinal cord, causing myelopathy and a radiculopathy. At lumbar levels, where a majority of disc herniations occur, the protruding nucleus pulposus does not produce a myelopathy because the spinal cord ends caudally at the level of the L1 vertebral body.

Spinal Cord: Gross Anatomy In Situ

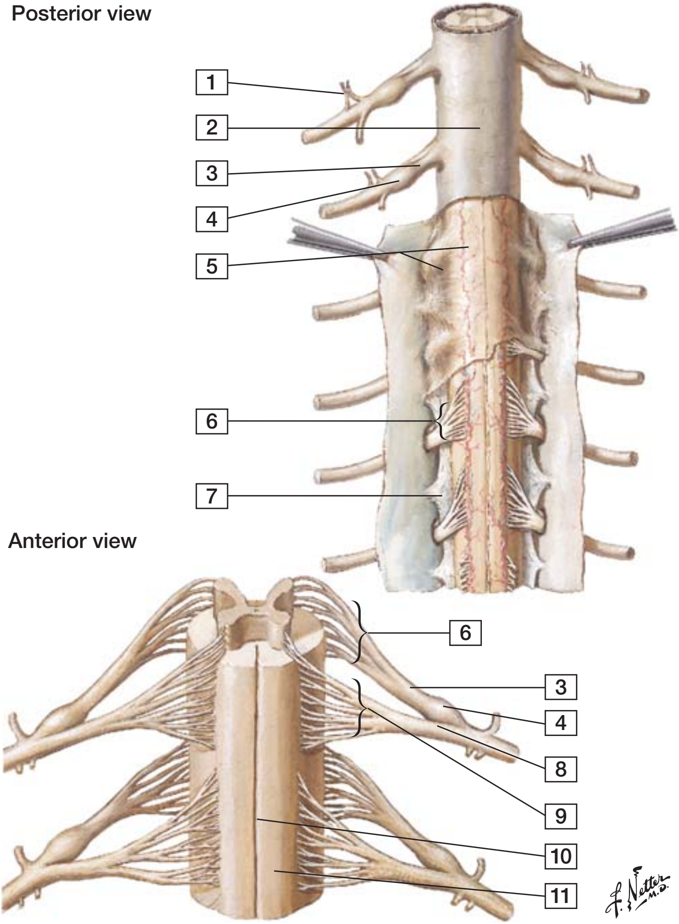


Spinal Cord: Gross Anatomy In Situ

1. First cervical nerve
2. Cervical enlargement
3. C7 nerve
4. Lumbosacral enlargement
5. L1 nerve
6. Conus medullaris
7. Cauda equina
8. L5 nerve
9. S1 nerve
10. Filum terminale

Comment: The cervical enlargement and lumbosacral enlargement of the spinal cord reflect the sensory inputs from the limbs and motor innervation to muscles of the limbs. During postnatal growth, the spinal column outstrips the spinal cord in longitudinal growth, resulting in the caudal end of the spinal cord (conus medullaris) ending at the L1 vertebral body level and the subsequent caudal stretching of the nerve roots to reach their entry/exit points through the intervertebral foramina. One clinically beneficial aspect of the spinal cord ending at the L1 vertebral level is the presence of a large subarachnoid space in the lumbar cistern from which cerebrospinal fluid can be withdrawn for analysis. The lumbar and sacral nerve roots course through the lumbar cistern but are not rigidly fixed and do not interfere with a lumbar puncture. Fractures of the lower spine, tumors, or other pathology can damage either the conus medullaris or cauda equina.

Spinal Cord: Its Meninges and Spinal Roots


Posterior view

Anterior view

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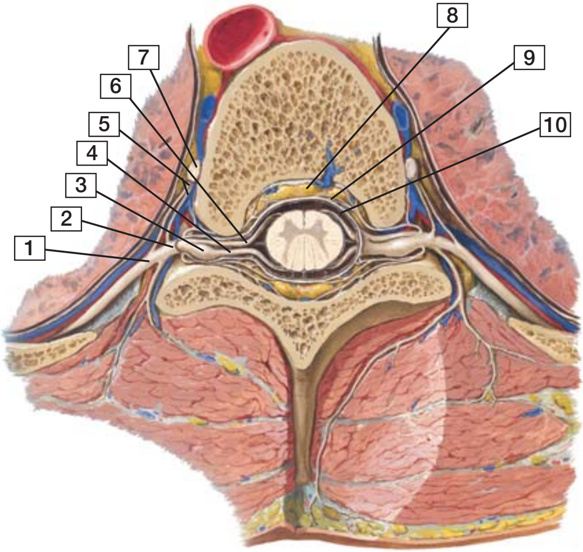
Spinal Cord: Its Meninges and Spinal Roots

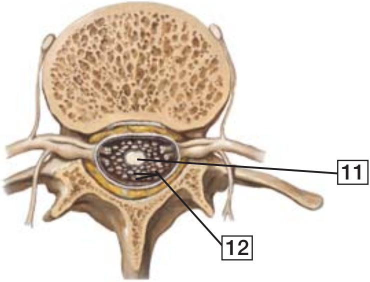
1. Rami communicantes
2. Dura mater
3. Dorsal root
4. Dorsal root (spinal) ganglion
5. Arachnoid membrane
6. Filaments of a dorsal root
7. Denticulate ligament
8. Ventral root
9. Filaments of a ventral root
10. Anterior median fissure
11. Anterior funiculus

Comment: Even though the dorsal and ventral roots emerge through the intervertebral foramina as 31 pairs of discrete nerve roots, a continuous line of small nerve rootlets coalesce to form these discrete nerve roots. The most vulnerable sites for herniated discs and subsequent nerve root compression are at L4-L5 and L5-S1, and less commonly the C6-C7, C5-C6, and C4-C5 cervical discs. Impingement of a herniated disc on a nerve root results in radiating pain in the specific distribution of that nerve root (a dermatomal pattern). However, the loss of sensation occurring from a nerve root lesion does not occur in a dermatomal pattern because of overlap of sensory projections to the spinal cord. At least three successive nerve roots would have to be damaged to produce complete anesthesia in a dermatomal pattern. This is not the case for the three divisions of the trigeminal nerve, which share no overlap.

A. Section through thoracic vertebra



B. Section through lumbar vertebra

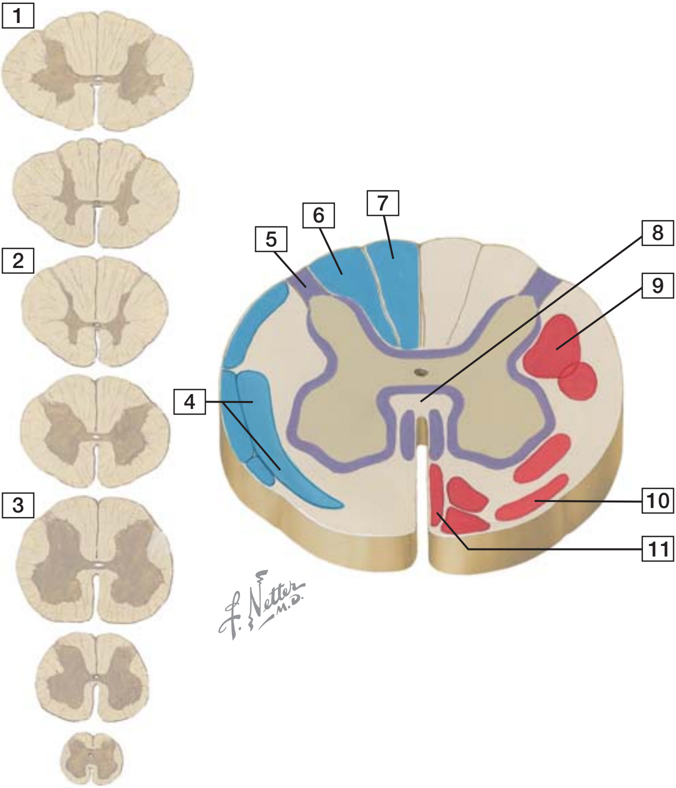


Spinal Cord: Cross-Sectional Anatomy In Situ

1. Ventral ramus (intercostal nerve)
2. Spinal nerve
3. Dorsal root (spinal) ganglion
4. Dorsal root
5. Rami communicantes
6. Ventral root
7. Sympathetic ganglion
8. Epidural space with epidural fat
9. Dura mater
10. Subarachnoid space
11. Filum terminale
12. Cauda equina

Comment: The dorsal root ganglia contain primary sensory cell bodies whose axons, when greater than 1 to 2 μm in diameter, are myelinated by Schwann cells in the peripheral nervous system, including the proximal portion that extends to the dorsal root entry zone of the spinal cord, and are myelinated by oligodendroglia once they enter the central nervous system. For thoracolumbar segments, the distal white ramus communicans contains axons from autonomic preganglionic neurons that will synapse in the sympathetic chain ganglia or continue through a splanchnic nerve to synapse in a collateral ganglion. The medially located gray ramus communicans contains the postganglionic axons (mostly unmyelinated) that distribute to target effector tissues of the autonomic nervous system. The bulk of the ventral root contains axons from lower motor neurons in the spinal cord, destined for synapsing on skeletal muscle fibers for that myotome.

Spinal Cord: White and Gray Matter

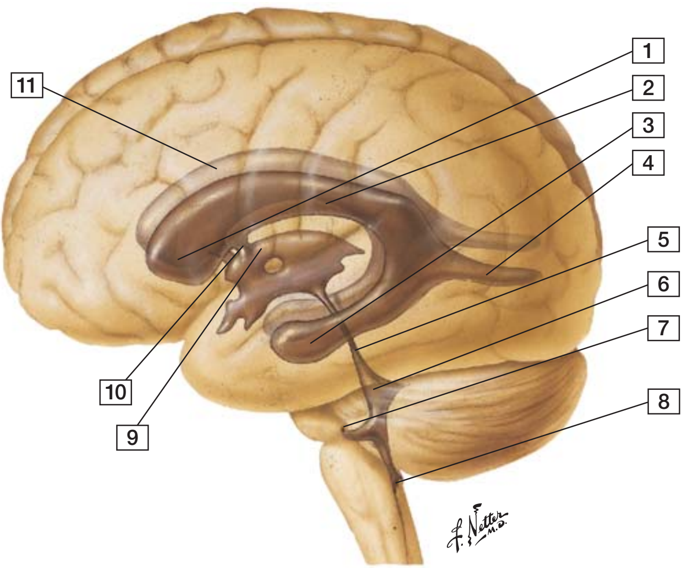


Spinal Cord: White and Gray Matter

1. C5 cross section
2. T8 cross section
3. L3 cross section
4. Spinothalamic/spinoreticular tracts
5. Dorsolateral fasciculus (of Lissauer)
6. Fasciculus cuneatus
7. Fasciculus gracilis
8. Anterior white commissure
9. Lateral corticospinal tract
10. Vestibulospinal tract
11. Anterior corticospinal tract

Comment: Spinal cord cross sections show variation in the size and distribution of gray matter and in the amount of white matter (axons). White matter increases in absolute amount from caudal to rostral. Gray matter shows enlargements of dorsal and ventral horns in the cervical and lumbosacral regions, reflecting inputs and outputs from the limbs. Gray matter is sparse in the thoracic region (body wall) and shows a conspicuous lateral horn (T1-L2) reflecting the intermediolateral cell column of preganglionic sympathetic neurons. The dorsal, lateral, and anterior funiculi consist of ascending and descending tracts subserving a wide range of functions. There is a separation of ascending tracts conveying fine, discriminative touch (epicritic sensation, primary sensory axons, dorsal funiculi) and pain and temperature sensation (protopathic sensation, secondary sensory axons, anterolateral funiculi). Descending motor axons of the lateral corticospinal and rubrospinal tracts travel in the lateral funiculus, and descending axons of the vestibulospinal and reticulospinal tracts travel in the anterior funiculus. Each white matter region may be damaged separately, reflected by specific sensory, motor, and autonomic symptoms.

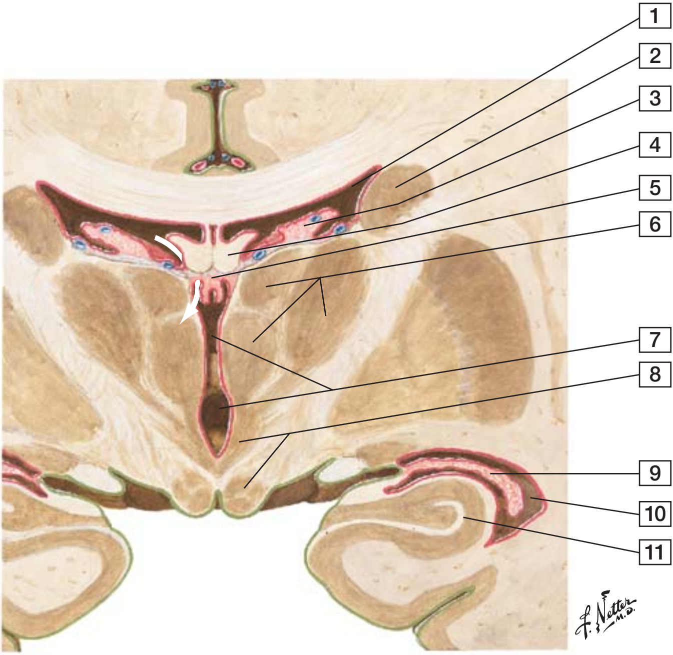
Ventricular Anatomy



Ventricular Anatomy

1. Anterior horn of left lateral ventricle
2. Body of left lateral ventricle
3. Inferior (temporal) horn of left lateral ventricle
4. Posterior (occipital) horn of left lateral ventricle
5. Cerebral aqueduct
6. Fourth ventricle
7. Left lateral foramen of Luschka
8. Median foramen of Magendie
9. Third ventricle
10. Left interventricular foramen of Monro
11. Right lateral ventricle

Comment: The third ventricle, cerebral aqueduct, and fourth ventricle developed from the embryonic central canal, responding to the waves of differential neuronal growth and neural tube folding that occurred progressively through development. Some regions (cerebral aqueduct) are narrow and are potential sites of obstruction of flow of cerebrospinal fluid internally (internal hydrocephalus). The lateral ventricles expanded in size and location into paired C-shaped structures, reflecting the growth of the forebrain in a sweeping C shape from the rostral portion of the neural tube. The lateral ventricles connect with the third ventricle through the interventricular foramen of Monro. The choroid plexus is present in the lateral, third, and fourth ventricles and produces the cerebrospinal fluid, which flows from rostral to caudal, escaping through the fourth ventricular apertures, the foramina of Luschka and of Magendie, into the subarachnoid space.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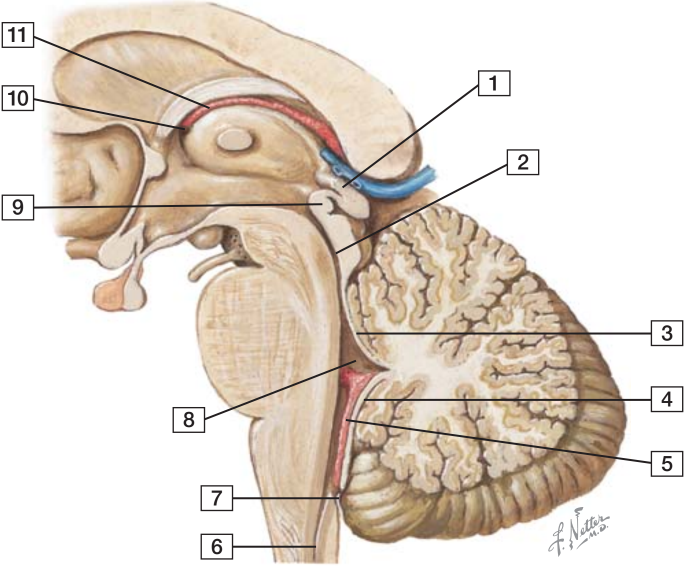
Ventricular Anatomy in Coronal Forebrain Section

1. Lateral ventricle
2. Body of caudate nucleus
3. Choroid plexus of lateral ventricle
4. Body of fornix
5. Choroid plexus of third ventricle
6. Thalamus
7. Third ventricle
8. Hypothalamus
9. Choroid plexus of lateral ventricle
10. Temporal horn of lateral ventricle
11. Hippocampus

Comment: A forebrain coronal section reveals two regions of the lateral ventricles, the body and the temporal horn. Cerebrospinal fluid flows from the lateral ventricles through the interventricular foramen of Monro into the narrow slitlike third ventricle. The regions adjacent to the walls and floor of the third ventricle in the hypothalamus are rich with neurons whose axons can secrete a variety of releasing factors, paracrine substances, and hormones. Some investigators consider the third ventricle as a site for fluid borne signals, derived from a variety of sources, to influence hypothalamic and neuroendocrine function.

Anatomy of the Fourth Ventricle: Lateral View



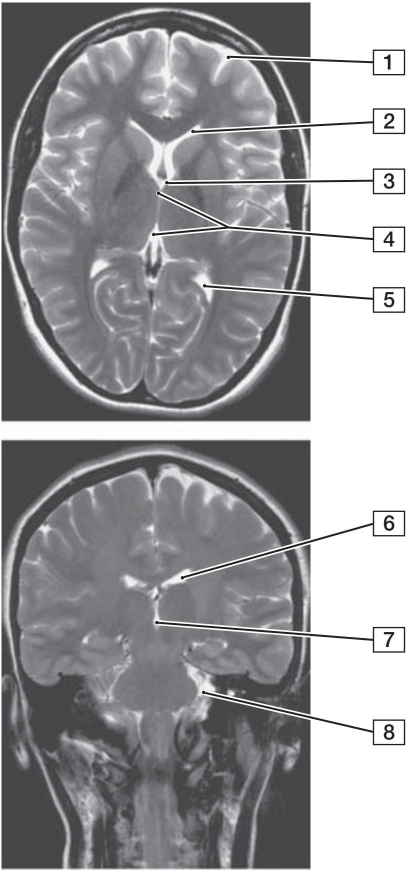
Anatomy of the Fourth Ventricle: Lateral View

1. Habenular commissure
2. Cerebral aqueduct
3. Superior medullary velum
4. Inferior medullary velum
5. Choroid plexus of fourth ventricle
6. Central canal of spinal cord
7. Median aperture (foramen of Magendie)
8. Fourth ventricle
9. Posterior commissure
10. Interventricular foramen of Monro
11. Choroid plexus of third ventricle

Comment: The fourth ventricle is a rhomboid-shaped structure in cross section, receiving cerebrospinal fluid from the cerebral aqueduct above and providing exit of CSF into the subarachnoid space through the paired lateral foramina of Luschka and the midline foramen of Magendie. Structurally, the roof and dorsolateral walls of the fourth ventricle are adjacent to the deep cerebellar nuclei and zones of the cerebellum itself. Just beneath the floor of the fourth ventricle are the tubercles, eminences, colliculi, and areas associated with the nuclei and associated connections of the cranial nerve nuclei. On the floor of the fourth ventricle is the sulcus limitans, the developmental demarcation of the alar plate laterally (sensory nuclei and structures) and the basal plate medially (motor and autonomic nuclei and structures).

Magnetic Resonance Imaging of the Ventricles: Axial and Coronal Views

A. Axial view

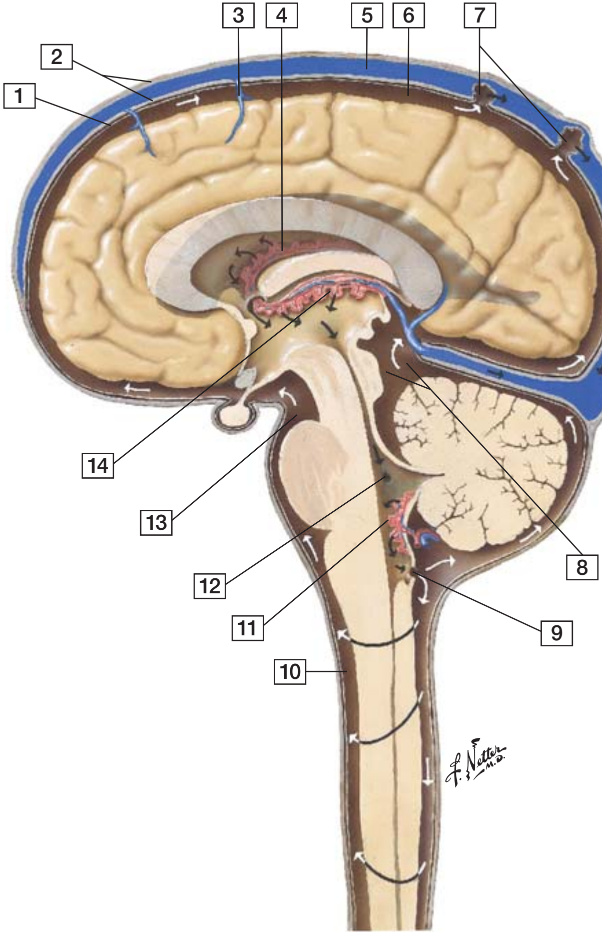


B. Coronal view

Magnetic Resonance Imaging of the Ventricles: Axial and Coronal Views

1. Subarachnoid space
2. Frontal pole of lateral ventricle
3. Interventricular foramen of Monro
4. Third ventricle
5. Temporal pole of lateral ventricle
6. Frontal pole of lateral ventricle
7. Third ventricle
8. Cisterns around brain stem

Comment: These T2-weighted magnetic resonance images show the ventricular system and the cisterns and subarachnoid space in white. The underlying anatomy of the head of the caudate nucleus and thalamus, separated by the internal capsule, is revealed. The cerebrospinal fluid is present in the subarachnoid space in all of the sulci and regions surrounding the brain, consistent with its important role in buoyancy and fluid protection of the fragile underlying brain tissue. Without such buoyancy, quick lateral or turning movements of the head could sheer long tracts. The subarachnoid space in the sulci can become obstructed by thick purulent exudates in acute purulent meningitis, preventing the smooth flow of cerebrospinal fluid from the subarachnoid space into the venous sinuses.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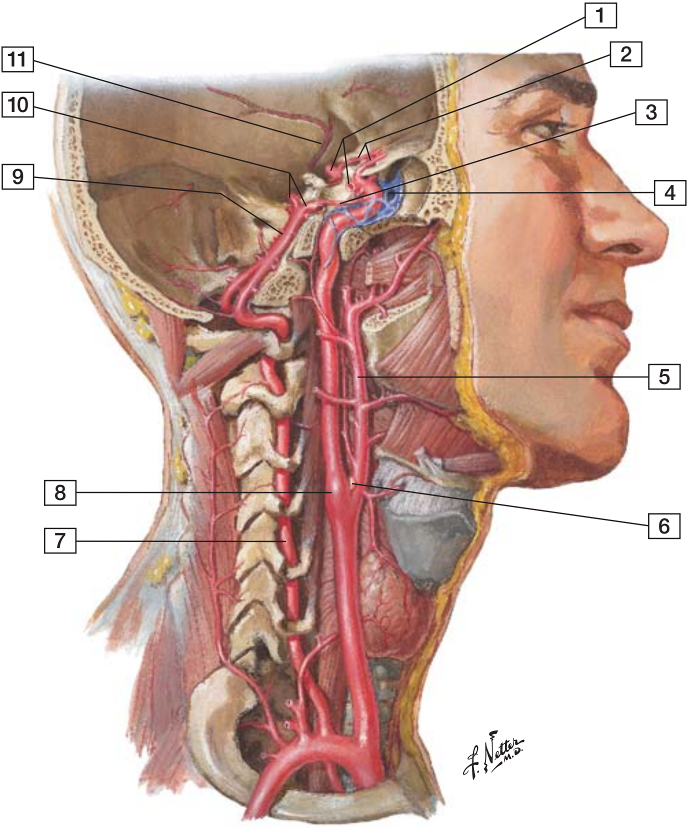
Circulation of the Cerebrospinal Fluid

1. Arachnoid membrane
2. Dura mater
3. Bridging veins
4. Choroid plexus of lateral ventricle
5. Superior sagittal sinus
6. Subarachnoid space
7. Arachnoid granulations
8. Cistern of the great cerebral vein
9. Median foramen of Magendie
10. Subarachnoid space
11. Choroid plexus of fourth ventricle
12. Lateral foramen of Luschka
13. Interpeduncular cistern
14. Choroid plexus of third ventricle

Comment: This classic Netter illustration of cerebrospinal fluid (CSF) flow from its origin in the choroids plexus, through the lateral ventricles, third ventricle, cerebral aqueduct, and fourth ventricle, into the subarachnoid space depicts its internal and external course of flow. The absorption of CSF occurs into the venous sinuses through the one-way valves, the arachnoid granulations, on the basis of pressure gradients. The cerebral arteries and veins travel in the subarachnoid space. The veins bridge into the sinuses and empty venous blood back into the general circulation via the sinuses and internal jugular vein. A ruptured berry aneurysm will result in a subarachnoid bleed, while torn bridging veins from trauma will dissect the dura and arachnoid, producing a subdural hematoma.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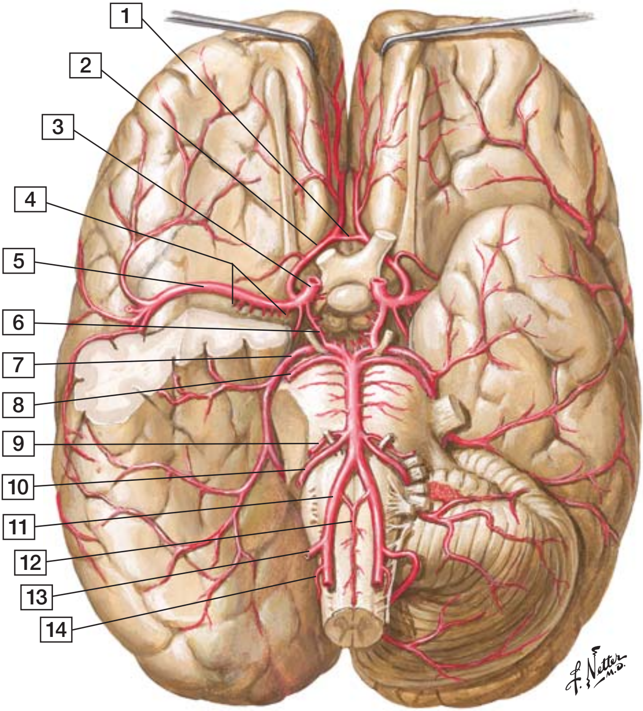
Arterial Supply to the Brain and Meninges

1. Right and left middle cerebral arteries
2. Right and left anterior cerebral arteries
3. Right posterior communicating artery
4. Cavernous sinus
5. Right internal carotid artery
6. Carotid body
7. Right vertebral artery
8. Right internal carotid artery
9. Basilar artery
10. Right and left posterior cerebral arteries
11. Left middle meningeal artery

Comment: The blood supply to the brain and rostral spinal cord derives from the paired internal carotid arteries (anterior circulation) and vertebral arteries (posterior circulation). The internal carotid arteries give rise to the anterior and middle cerebral arteries, supplying most of the forebrain with blood except for the occipital lobe and inferior portion of the temporal lobe. The two vertebral arteries unite to form the basilar artery, whose end cerebral branches are the posterior cerebral arteries, supplying the occipital lobe and inferior portion of the temporal lobe. The vertebrobasilar system also supplies the brain stem, cerebellum, and rostral (cervical) spinal cord with blood. The cerebral arteries and their branches are mainly end arteries, with little margin for anastomotic channels; thus, an occlusion will usually damage tissue supplies with blood downstream to the occlusion. The brain has little energy reserve and depends moment to moment on delivery of oxygen and glucose for robust aerobic metabolism for neural tissue. The bifurcation of the common carotid artery is a common site of atherosclerotic plaque formation. The circle of Willis, the site of communicating artery interconnections between the anterior and posterior circulation, is a common site of berry aneurysms.

Arterial Distribution to the Brain: Basal View

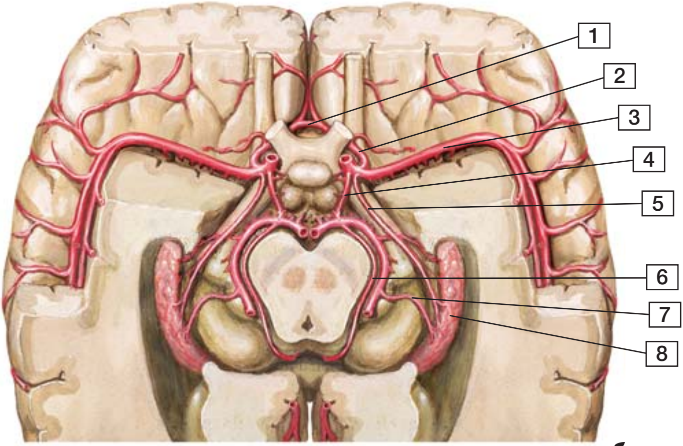


Arterial Distribution to the Brain: Basal View

1. Anterior communicating artery
2. Anterior cerebral artery
3. Internal carotid artery
4. Medial and lateral lenticulostriate arteries
5. Middle cerebral artery
6. Posterior communicating artery
7. Posterior cerebral artery
8. Superior cerebellar artery
9. Internal auditory (labyrinthine) artery
10. Anterior inferior cerebellar artery
11. Vertebral artery
12. Anterior spinal artery
13. Posterior inferior cerebellar artery
14. Posterior spinal artery

Comment: The vertebrobasilar circulation supplies posterior circulation to the brain stem and cerebellum, part of the diencephalon, the occipital lobe, and inferior part of the temporal lobe. The vertebral arteries and basilar artery give rise to paramedian, short circumferential, and long circumferential arteries that supply wedge-shaped territories or specific medial and lateral territories of the brain stem and cerebellum with blood. An occlusion may lead to damage of cranial nerve and brain stem nuclei, as well as long tracts, leading to highly predictable but unusual clusters of symptoms such as the lateral medullary syndrome. The middle and anterior cerebral arteries give rise to specific named branches that supply blood to specific regions of the hemisphere. Disruption can result in symptoms specific to the role subserved by the cortical or subcortical regions affected.

Arterial Distribution to the Brain: Cutaway Basal View Showing the Circle of Willis



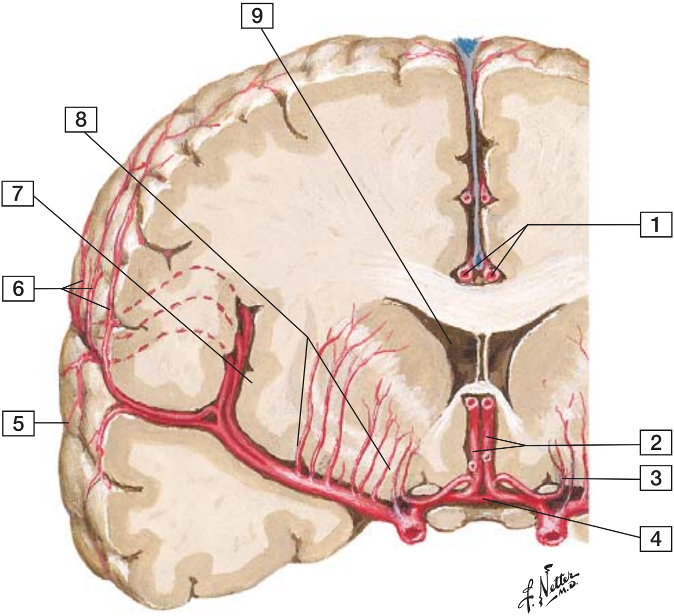
F. Netter M.D.
with
J. Perkins
MS, MFA

Arterial Distribution to the Brain: Cutaway Basal View Showing the Circle of Willis

1. Anterior communicating artery
2. Anterior cerebral artery
3. Middle cerebral artery
4. Posterior communicating artery
5. Anterior choroidal artery
6. Posterior medial choroidal artery
7. Posterior lateral choroidal artery
8. Choroid plexus of lateral ventricle

Comment: The circle of Willis is an anatomical interconnection of nine arteries that structurally bring the anterior and posterior circulation together. The paired posterior communicating arteries interconnect the posterior cerebral and middle cerebral arteries. The anterior communicating artery interconnects the paired anterior cerebral arteries. The size and patency of the communicating arteries varies considerably. The free flow of blood through the communicating arteries is usually insufficient to perfuse the brain adequately in the face of an occlusion to a major cerebral artery. Full patency of blood flow between the anterior and posterior circulations through the circle of Willis occurs only in approximately 20% of the population.

Arterial Distribution to the Brain: Coronal Forebrain Section

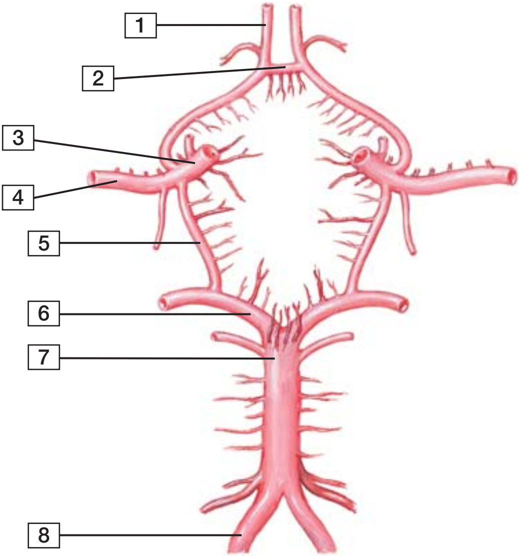


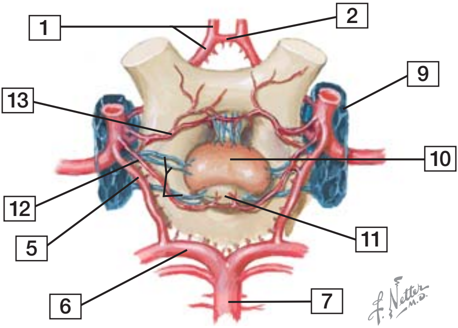
Arterial Distribution to the Brain: Coronal Forebrain Section

1. Pericallosal branches of anterior cerebral artery
2. Anterior cerebral arteries
3. Recurrent artery of Heubner
4. Anterior communicating artery
5. Temporal branches of middle cerebral artery
6. Precentral, central, and parietal branches of middle cerebral artery
7. Insula
8. Medial and lateral lenticulostriate arteries
9. Frontal pole (horn) of lateral ventricle

Comment: The lenticulostriate arteries are thin, penetrating branches of the middle cerebral artery (MCA) that arise shortly after the MCA branches from the internal carotid artery. These arteries penetrate into the basal ganglia and internal capsule; they are a frequent site of cerebral infarcts and have been called the "arteries of stroke." A stroke affecting these arteries can produce a classical contralateral hemiplegia with hypertonus and hyperreflexia, often affecting the upper extremity more severely than other regions. Such a stroke on the left may be accompanied by expressive aphasia if the stroke extends distally.

Circle of Willis: Schematic Illustration and Vessels In Situ



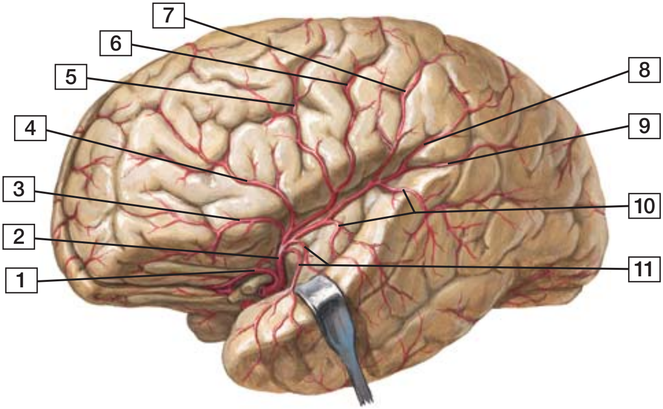


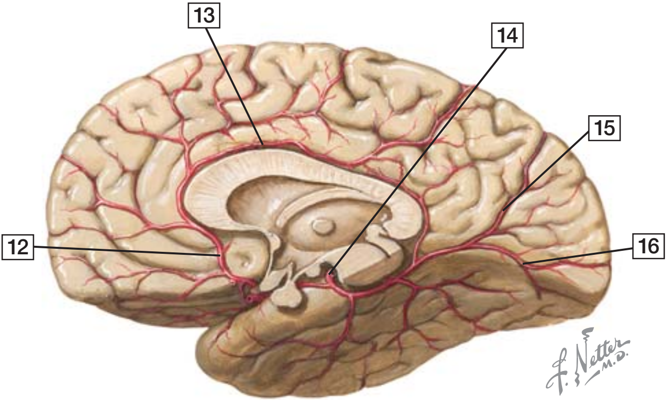
Circle of Willis: Schematic Illustration and Vessels In Situ

1. Anterior cerebral artery
2. Anterior communicating artery
3. Internal carotid artery
4. Middle cerebral artery
5. Posterior communicating artery
6. Posterior cerebral artery
7. Basilar artery
8. Vertebral artery
9. Cavernous sinus
10. Adenohypophysis (anterior pituitary gland)
11. Neurohypophysis (posterior pituitary gland)
12. Inferior hypophyseal artery
13. Superior hypophyseal artery

Comment: The circle of Willis consists of nine arteries that interconnect the anterior and posterior circulations (paired anterior, middle, and posterior cerebrals; paired posterior communicating arteries; single anterior communicating artery). The communicating arteries and the major cerebral arteries also give rise to many small penetrating or perforating arteries that supply adjacent thalamus, hypothalamus, and other structures. Of particular importance is the blood supply to the hypothalamus, median eminence, and pituitary, sites where neuroendocrine transduction occurs. Saccular (berry) aneurysms are outpouchings of cerebral arteries that represent weakness in the wall; they may rupture and bleed into the subarachnoid space, producing acute meningeal signs, headache, nausea and vomiting, possible loss of consciousness, and even death.

Arterial Distribution to the Brain: Lateral and Medial Views



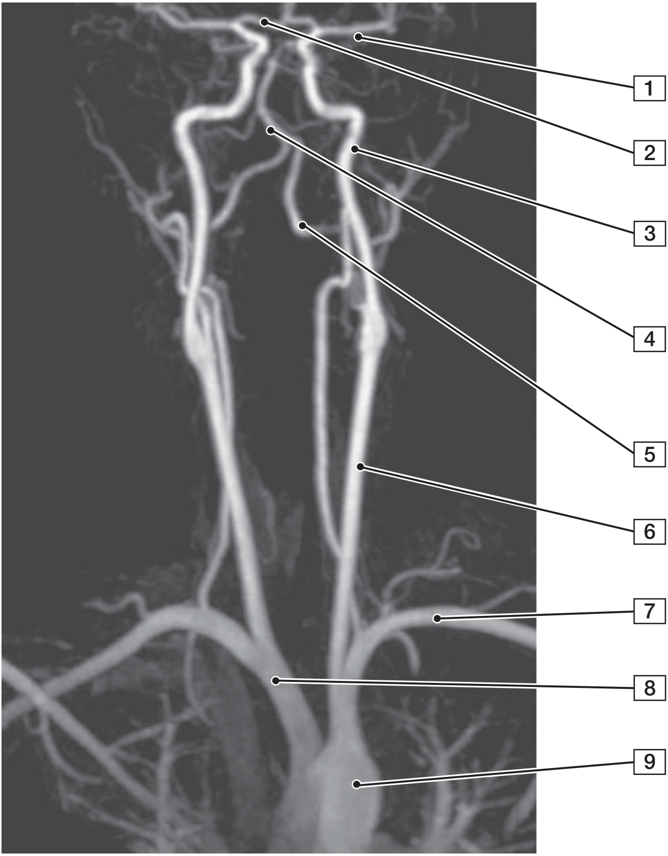


Arterial Distribution to the Brain: Lateral and Medial Views

1. Left anterior cerebral artery
2. Left middle cerebral artery (MCA)
3. Lateral orbitofrontal branch of MCA
4. Ascending frontal (candelabra) branch of MCA
5. Precentral branch of MCA
6. Central branch of MCA
7. Anterior parietal branch of MCA
8. Posterior parietal branch of MCA
9. Angular branch of MCA
10. Posterior temporal branches of MCA
11. Anterior temporal branches of MCA
12. Right anterior cerebral artery (ACA)
13. Pericallosal branch of ACA
14. Right posterior cerebral artery (PCA)
15. Parietooccipital branch of PCA
16. Calcarine branch of PCA

Comment: Individual arterial branches of the ACA, MCA, and PCA supply specific territories of the hemisphere with their blood supply. These branches are subject to strokes (1/3 embolic, 1/3 atherosclerotic, 1/5 lacunar). These strokes can affect regions of the cerebral cortex, basal ganglia, internal capsule, thalamus, and cerebral white matter.

Magnetic Resonance Angiography: Coronal Full Vessel View

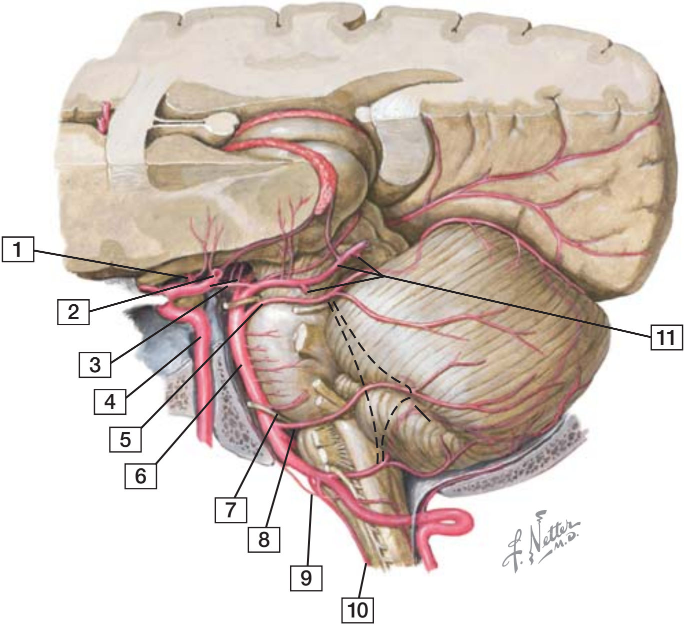


Magnetic Resonance Angiography: Coronal Full Vessel View

1. Middle cerebral artery
2. Anterior cerebral artery
3. Internal carotid artery
4. Basilar artery
5. Vertebral artery
6. Common carotid artery
7. Subclavian artery
8. Brachiocephalic trunk
9. Aortic arch

Comment: Magnetic resonance angiography takes advantage of the properties of macroscopic blood flow to render images of cerebral vessels. The blood signal can be made to appear either dark or bright. This technique has replaced most of the need for cerebral angiography based on injection of radiopaque contrast agents. Classical angiography with radiopaque contrast agents can produce extraordinarily detailed anatomical depiction of fine arterial branches, but magnetic resonance angiography can provide excellent visualization of arteries for neurological diagnostic purposes.

See book 7.13, C only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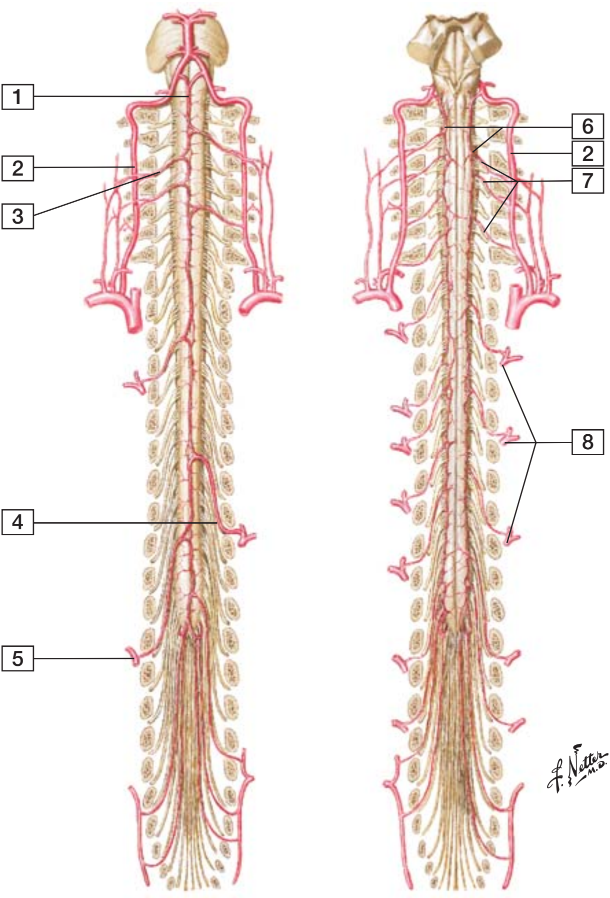
Vertebrobasilar Arterial System

1. Anterior cerebral artery
2. Middle cerebral artery
3. Posterior communicating artery
4. Left internal carotid artery
5. Superior cerebellar artery
6. Basilar artery
7. Internal auditory (labyrinthine) artery
8. Anterior inferior cerebellar artery
9. Anterior meningeal branch of vertebral artery
10. Anterior spinal artery
11. Left posterior cerebral artery

Comment: Following a hairpin turn before penetrating through the dura, the vertebral arteries travel along the anterior surface of the medulla and unite to form the basilar artery in the basilar groove on the anterior surface of the pons. Paramedian penetrating branches supply a medial wedge of medullary or pontine tissue with blood. An infarct in these penetrating arteries results in ipsilateral cranial nerve damage (e.g., ipsilateral paresis and wasting of intrinsic muscles of the tongue with damage to CN XII) and contralateral motor damage (contralateral hemiparesis, corticospinal tract) and somatosensory damage (loss of fine discriminative touch and epicritic sensations, medial lemniscus). This complex of symptoms is referred to as an *alternating hemiplegia*. An infarct in a circumferential artery results in a complex array of sensory, motor, and autonomic dysfunction based on local damage to brain stem nuclei and to damage of passing sensory, motor, and autonomic long tracts.

Arterial Blood Supply to the Spinal Cord: Longitudinal View

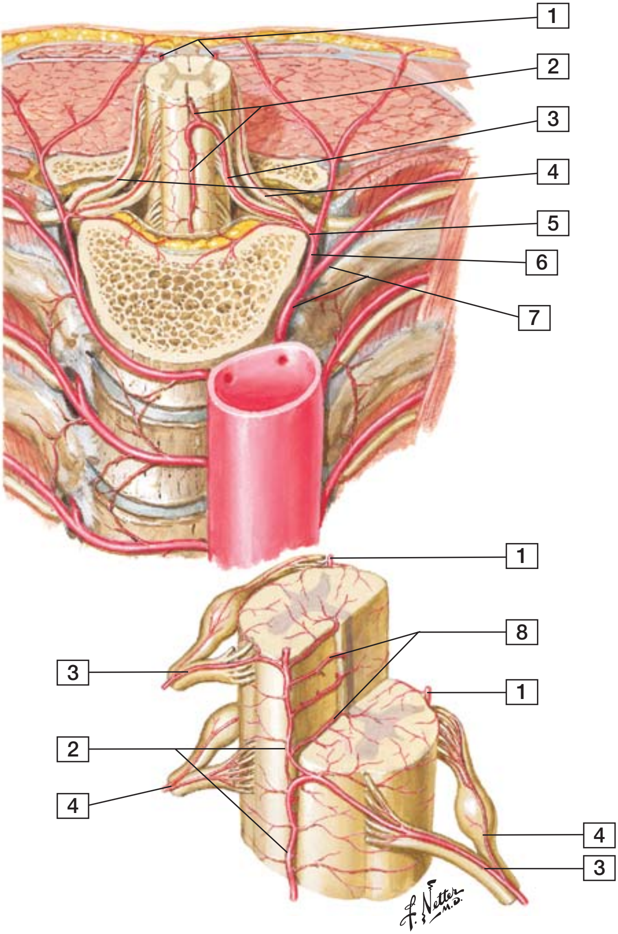


Arterial Blood Supply to the Spinal Cord: Longitudinal View

1. Anterior spinal artery
2. Vertebral artery
3. Anterior radicular artery
4. Artery of Adamkiewicz (radicular artery)
5. Lumbar artery
6. Posterior spinal arteries
7. Posterior radicular arteries
8. Posterior intercostal arteries

Comment: Both the anterior spinal artery and the paired posterior spinal arteries arise from the vertebral arteries and appear to descend along the entire course of the spinal cord, the anterior spinal artery in the anterior median fissure, and the posterior spinal arteries medial to the dorsal root entry zone. However, the force of blood flow through these arteries, based on vertebral blood supply, is insufficient to keep more than the cervical segments fully supplied with blood. Thus, the anterior and posterior spinal arteries need supplemental blood flow from radicular arteries that derive from intercostal arteries, branching from the descending aortic flow. Impaired supplemental blood flow from these vital radicular arteries can result in spinal cord infarct, an important point for surgeons to note when it is necessary to acutely shut off the blood flow through the descending aorta for a period of time.

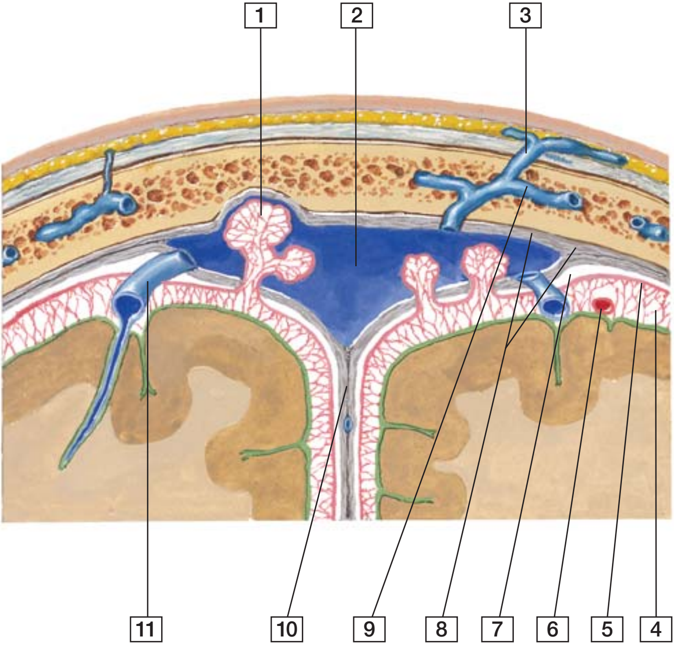
Arterial Supply to the Spinal Cord: Cross-Sectional View



Arterial Supply to the Spinal Cord: Cross-Sectional View

1. Posterior spinal arteries
2. Anterior spinal artery
3. Anterior radicular artery
4. Posterior radicular arteries
5. Spinal branch of dorsal ramus of posterior intercostal artery
6. Dorsal ramus of posterior intercostal artery
7. Posterior intercostal arteries
8. Central branches to left side of the spinal cord

Comment: The anterior spinal artery supplies the anterior two thirds of the spinal cord with blood. Full occlusion of this artery results in local damage to lower motor neurons (flaccid paralysis of innervated muscles with loss of tone and reflexes) in the affected territory and spastic quadriparesis or paraparesis, depending on the level, from disruption of descending corticospinal and rubrospinal tracts in the lateral funiculus, affecting muscles for which the lower motor neurons are still intact and functional. Damage to the lateral funiculus produces loss of pain and temperature sensation (anterolateral funiculus) and loss of bowel and bladder control (disruption of descending autonomic fibers in the lateral funiculus).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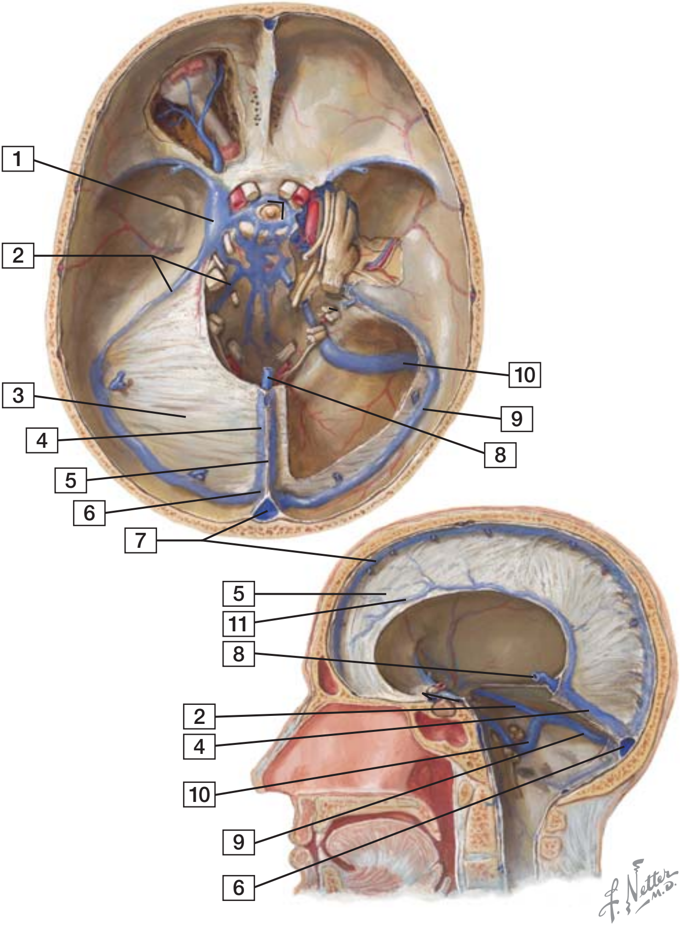
Meninges and Superficial Cerebral Veins

1. Arachnoid granulation
2. Superior sagittal sinus
3. Emissary vein
4. Subarachnoid space
5. Arachnoid membrane
6. Cerebral artery
7. Subdural space (potential space)
8. Dura mater
9. Diploid vein
10. Falx cerebri
11. Bridging cerebral vein

Comment: Cerebral arteries and veins travel in the subarachnoid space. The bridging veins from the cortical surface penetrate through the arachnoid and inner layer of the dura to empty into the venous sinuses. Arachnoid granulations also penetrate through the arachnoid and inner layer of the dura to convey cerebrospinal fluid from the subarachnoid space into the venous sinuses. Additional veins from the skull (diploic vein) and from surface regions that penetrate the galea aponeurotica (emissary veins) also empty into the venous sinuses. These additional veins can be sources that convey infectious organisms into the cerebral venous drainage from outside the cranium.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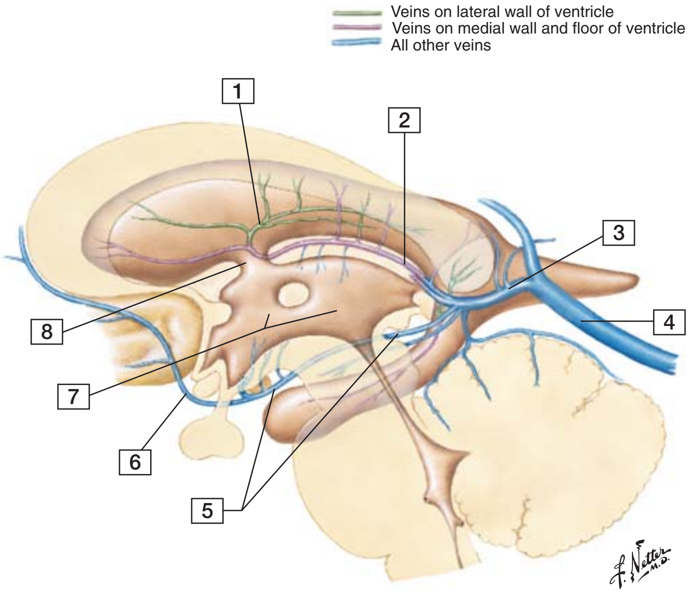
Venous Sinuses

1. Cavernous sinus
2. Superior and inferior petrosal sinuses
3. Tentorium cerebelli
4. Straight sinus
5. Falx cerebri
6. Confluence of sinuses
7. Superior sagittal sinus
8. Great cerebral vein (of Galen)
9. Transverse sinus
10. Sigmoid sinus
11. Inferior sagittal sinus

Comment: Venous sinuses are channels formed by a split of the dura (inner and outer layers) that drain blood from the brain, meninges, and the skull, and they also drain cerebrospinal fluid from the subarachnoid space via the arachnoid granulations. The veins that drain into the sinuses are without valves and can convey infections (e.g., from the paranasal sinuses or middle ear) into the sinuses (cavernous sinus). The inferior sagittal sinus and great cerebral vein drain into the straight sinus. The straight sinus and superior sagittal sinus converge at the confluence of sinuses and continue the combined drainage through the transverse sinuses and sigmoid sinuses into the internal jugular vein.

Deep Venous Drainage of the Brain: Relationship to the Ventricles


Veins on lateral wall of ventricle
Veins on medial wall and floor of ventricle
All other veins
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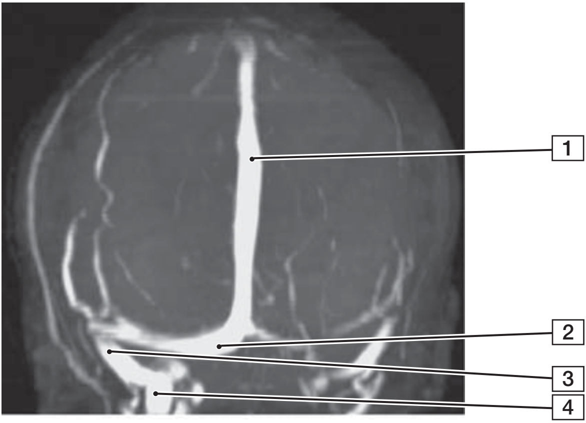
Deep Venous Drainage of the Brain: Relationship to the Ventricles

1. Thalamostriate vein
2. Internal cerebral vein
3. Great cerebral vein of Galen
4. Straight sinus
5. Basal vein of Rosenthal
6. Anterior cerebral vein
7. Third ventricle
8. Interventricular foramen of Monro

Comment: The great cerebral vein of Galen is a site of convergence of venous drainage from the anterior cerebral vein and basal vein of Rosenthal and its branches, the posterior mesencephalic vein, cerebellar veins, the internal cerebral vein and its branches (anterior septal vein, thalamostriate vein), atrial veins, pericallosal veins, and others. The great cerebral vein and inferior sagittal sinus unite to form the straight sinus. Many of these draining veins follow the contours of the ventricles as they converge toward the great cerebral vein of Galen. Blockage or occlusion of a vein can result in backup of perfusion with resultant ischemia of the tissue in the region of drainage.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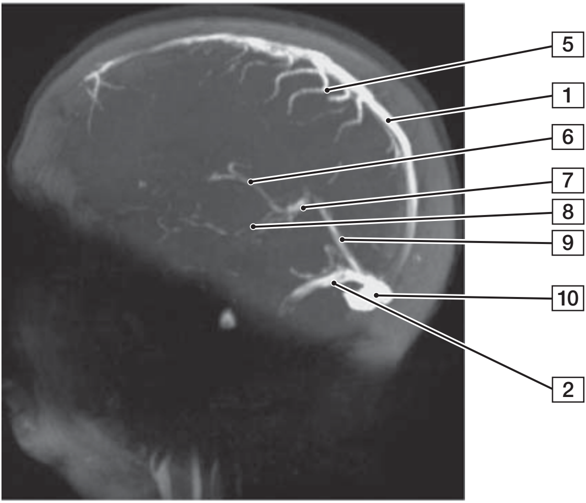
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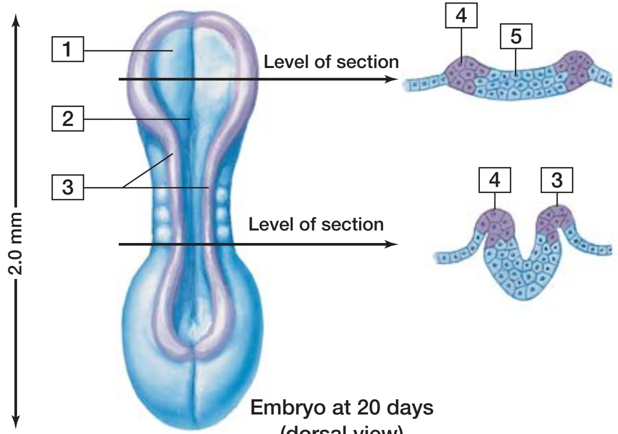
Magnetic Resonance Venography

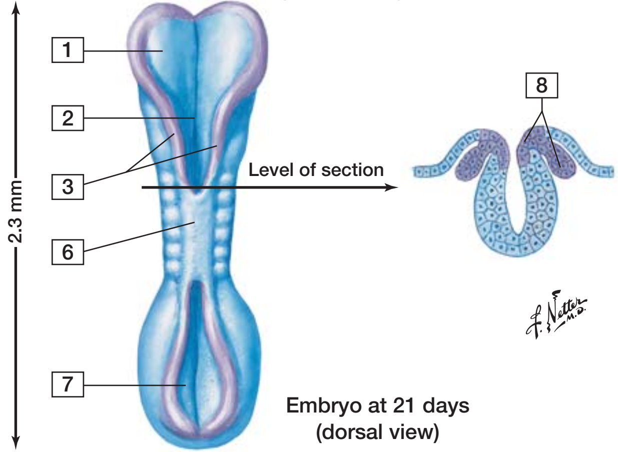
1. Superior sagittal sinus
2. Transverse sinus
3. Sigmoid sinus
4. Internal jugular vein
5. Cerebral veins
6. Internal cerebral vein
7. Great cerebral vein of Galen
8. Basal vein of Rosenthal
9. Straight sinus
10. Confluence of sinuses

Comment: Magnetic resonance (MR) venography uses the same principles of flow as MR angiography, taking advantage of the relatively slow and steady venous blood flow compared with the more robust flow of cerebral arterial blood. MR venography has largely replaced imaging of venous drainage with radiopaque contrast agents from carotid arteriography (venous phase).

Neurulation



Embryo at 20 days
(dorsal view)



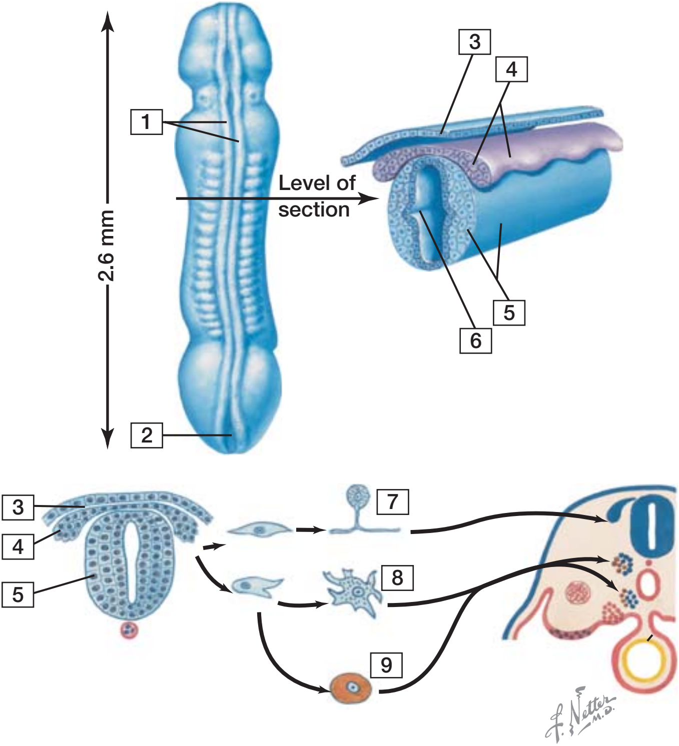
Embryo at 21 days
(dorsal view)

Neurulation

1. Neural plate of forebrain
2. Neural groove
3. Neural folds
4. Future neural crest
5. Neural plate
6. Fused neural folds
7. Caudal neuropore
8. Neural crest

Comment: The neural plate invaginates and forms the neural tube as the two margins of the neural folds fuse. This process of neurulation starts centrally and moves both caudally and rostrally. If the neural folds fail to fuse properly, dysraphic effects are seen that involve altered development of associated muscles, bone, skin, and meninges. The failure of the caudal neuropore to close results in spina bifida, with failure of vertebral arches to fuse and possible protrusion of meninges and neural tissues, exposing them to the external environment. If the rostral neuropore fails to form, anencephaly results, a failure of brain development with exposure of the neural rudiments to the external environment, a lethal condition.

Neural Tube Development and Neural Crest Formation

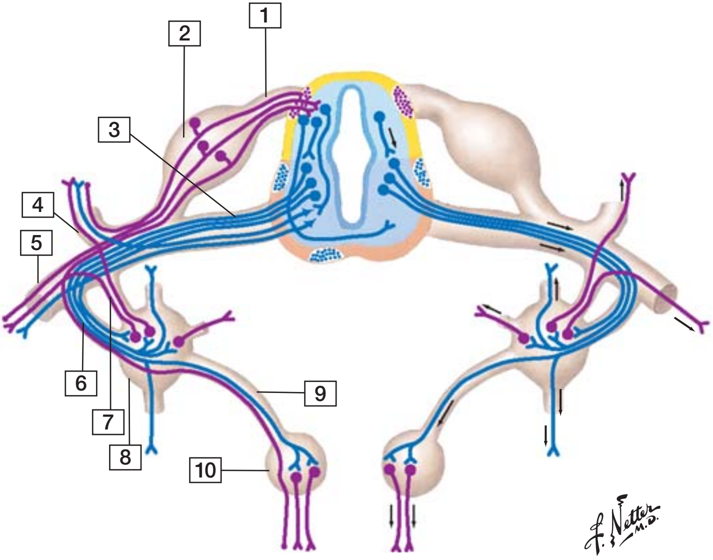


Neural Tube Development and Neural Crest Formation

1. Fused neural folds
2. Caudal neuropore
3. Ectoderm
4. Neural crest
5. Neural tube
6. Sulcus limitans
7. Sensory neurons of dorsal root (spinal) ganglion
8. Visceral motor (autonomic) neuron of sympathetic ganglion
9. Chromaffin cells of adrenal medulla

Comment: As the neural folds fuse to form the neural tube, developing neural crest develops dorsal to the neural tube. Cells of the neural crest migrate outward and ventrally to give rise to a wide range of cells, including primary sensory neurons, ganglion cells of the autonomic nervous system, adrenal medullary chromaffin cells, Schwann cells, pia and arachnoid membrane cells, melanocytes, and some mesenchyme of the head. Failure of neural crest cells to properly migrate and develop can result in disorders such as congenital megacolon (Hirschsprung's disease) where sensory signaling from the colon is absent, or familial dysautonomia, in which autonomic symptoms (cardiovascular, gastrointestinal) and sensory deficits (pain and temperature sensation) are seen.

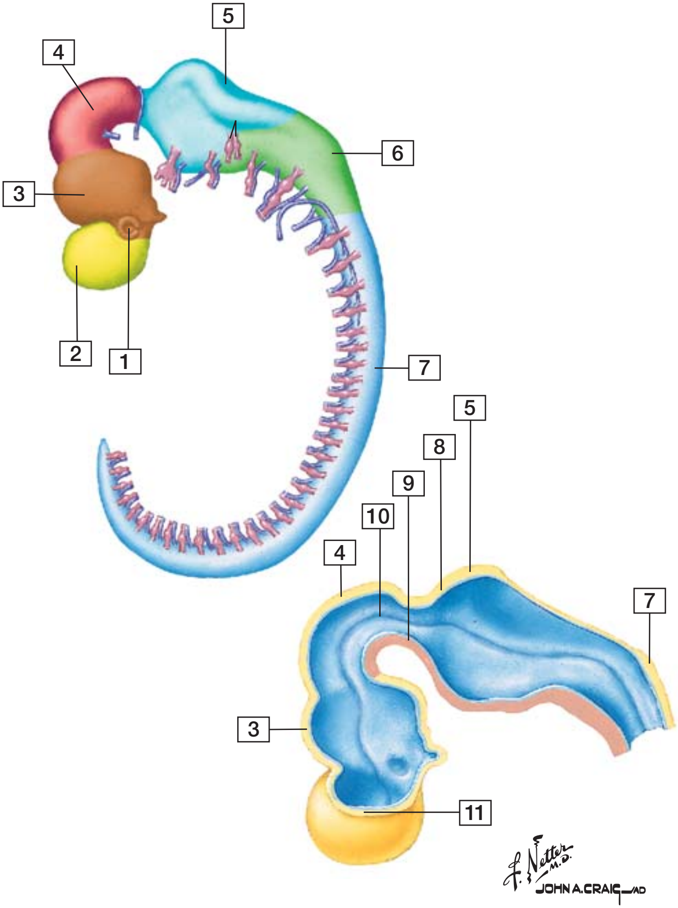
Development of Peripheral Axons



Development of Peripheral Axons

1. Dorsal (sensory) root
2. Dorsal (sensory) ganglion
3. Ventral (motor) root
4. Dorsal ramus of spinal nerve
5. Ventral ramus of spinal nerve
6. White ramus communicans
7. Gray ramus communicans
8. Sympathetic chain (trunk) ganglion
9. Splanchnic nerve
10. Sympathetic collateral (trunk) ganglion

Comment: Neural crest cells give rise to (1) dorsal root ganglia and primary sensory cranial nerve ganglia, as well as their pseudounipolar distal and proximal axons; (2) sympathetic chain ganglia and their postganglionic axons; (3) sympathetic collateral ganglia and their postganglionic axons, located near the organs innervated; and (4) parasympathetic ganglion cells and their postganglionic axons, located near the organs innervated. All preganglionic autonomic neurons and their axons (including preganglionic axons in splanchnic nerves) derive from the neural tube. All lower motor neurons and their axons also derive from the neural tube.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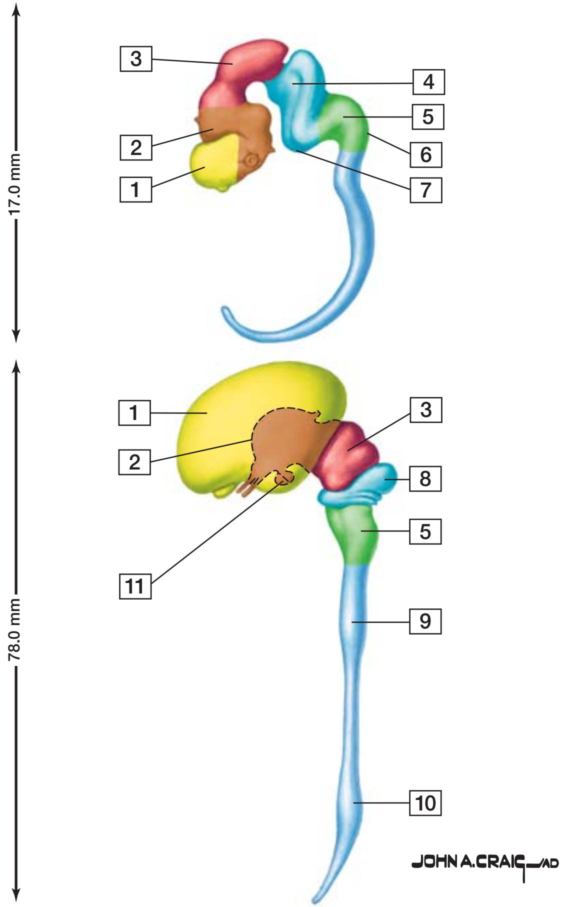
Early Brain Development: 36-Day-Old Embryo

1. Optic cup
2. Telencephalic vesicle
3. Diencephalon
4. Midbrain (mesencephalon)
5. Hindbrain (metencephalon)
6. Hindbrain (myelencephalon)
7. Spinal cord
8. Alar plate
9. Basal plate
10. Sulcus limitans
11. Lamina terminalis

Comment: As the neural tube grows and develops rostrally, neural proliferation and migration occurs in differential fashion; this leads to multiple folds that help to demarcate the spinal cord from the brain stem (cervical flexure), the medulla and pons in the rhombencephalon (pontine flexure), and the mesencephalon from the prosencephalon (cephalic flexure). This process of neural growth and folding also affects the ventricular system, which goes from a simple tube (central canal) to a complex system of ventricles, including the rhomboid fourth ventricle (rhombencephalon), small cerebral aqueduct (mesencephalon), third ventricle (diencephalon), and lateral ventricles (telencephalon).

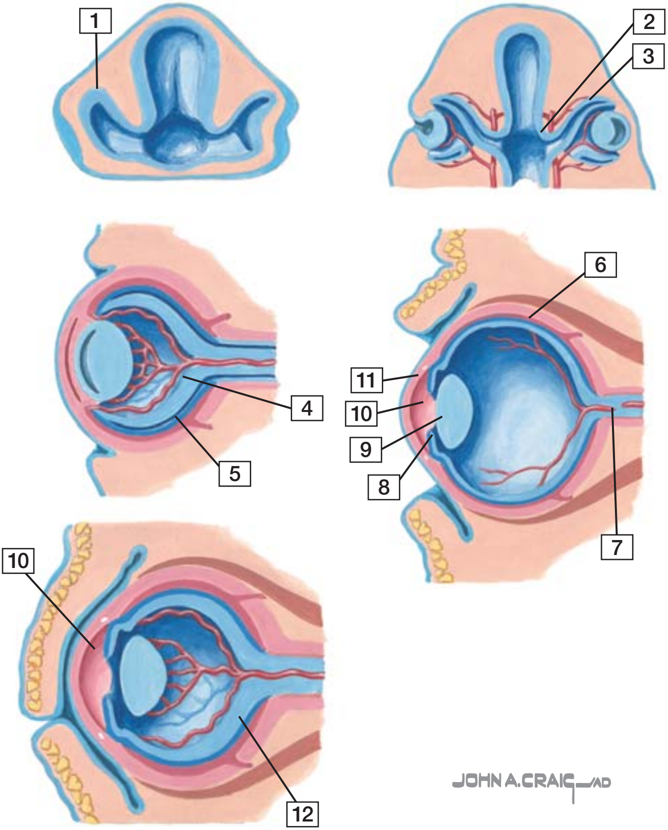
Early Brain Development: 49-Day-Old Embryo and 3-Month-Old Embryo



Early Brain Development: 49-Day-Old Embryo and 3-Month-Old Embryo

1. Telencephalic vesicle (cerebral hemispheres, neocortex)
2. Diencephalon
3. Mesencephalon
4. Metencephalon (cerebellum, pons)
5. Myelencephalon (medulla oblongata)
6. Cervical flexure
7. Pontine flexure
8. Cerebellum
9. Cervical enlargement of spinal cord
10. Lumbosacral enlargement of spinal cord
11. Hypophysis (pituitary gland)

Comment: From 49 days to 3 months, the telencephalon undergoes massive growth and development. The telencephalon (cerebral cortex, basal ganglia, limbic forebrain, olfactory structures) expands around the diencephalon and overgrows it (encephalization). In the brain stem, the superior and inferior colliculi expand from the dorsal surface of the mesencephalon, and the cerebellum expands from the rhombic lips of the fourth ventricle in the metencephalon.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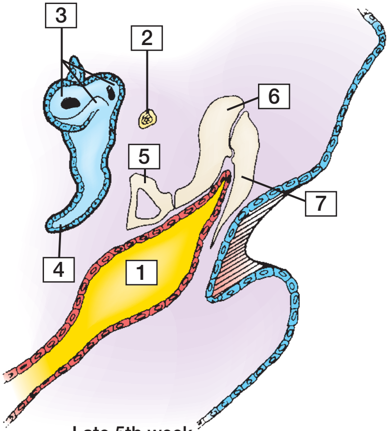
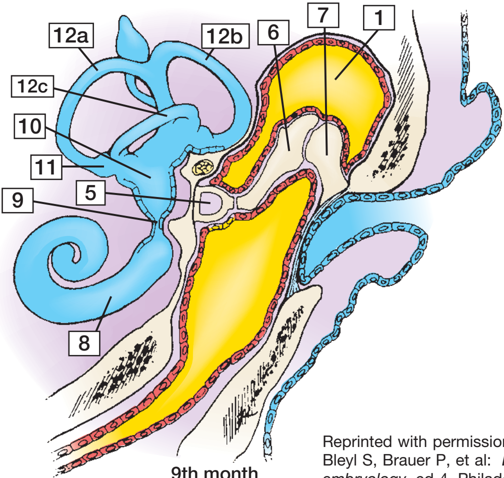
Development of the Eye and Orbit

1. Optic vesicle
2. Optic stalk
3. Optic cup
4. Inner layer of optic vesicle (visual retina)
5. Outer layer of optic vesicle (pigmented retinal epithelium)
6. Choroid (mesenchyme)
7. Optic nerve (neuroectoderm)
8. Iris (neuroectoderm)
9. Lens (surface ectoderm)
10. Anterior chamber
11. Cornea (mesenchyme)
12. Fusion of visual retina and pigmented retinal epithelium

Comment: The retina and optic nerve develop as an extension of the neural tube (diencephalon). The optic cup expands from the diencephalon as a two-layered structure; the two layers fuse but remain a site of possible separation in retinal detachment. The retina itself is neuroectodermal in origin and part of the central nervous system. The retinal neurons, including the ganglion cells and their optic nerve fibers (myelinated by oligodendroglia), are central neural structures and their vascular supply is central neural vasculature, the only central neural and vascular structures that can be visualized directly by ophthalmoscopy. The iris and ciliary body also are derived in part from neuroectodermal derivatives from the optic cup.



Late 5th week



9th month

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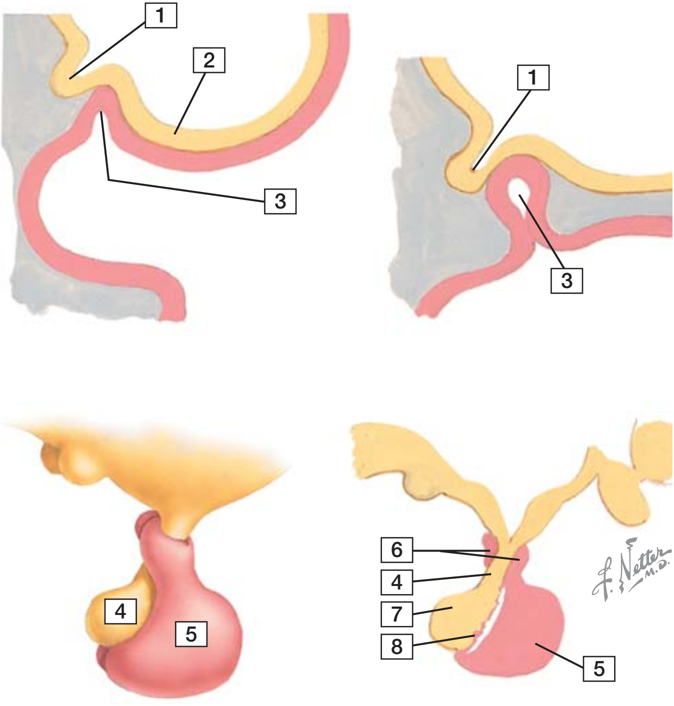
Development of the Ear

1. Tympanic cavity
2. Cranial nerve (CN) VIII
3. Developing semicircular canals
4. Developing cochlear duct
5. Stapes
6. Incus
7. Malleus
8. Cochlear duct
9. Ductus reuniens
10. Utricle
11. Ampulla
12. Semicircular canals (12a—anterior; 12b—posterior; 12c—lateral)

Comment: CN VIII derives from neural crest cells. The outer ear (auricle, external auditory meatus) develops from the first pharyngeal grove; the middle ear (the ossicles) develops from the first pharyngeal pouch; and the inner ear (bony labyrinth, membranous labyrinth, the cochlea, the semicircular canals) develops from the otic placode. Hair cells associated with the utricle (otolith organ) and semicircular canals (ampullae) respond to linear acceleration and angular acceleration, respectively. Hair cells in the organ of Corti on the basilar membrane respond to differential movement of the tectorial membrane and basilar membrane due to fluid waves generated through the scala vestibule and tympani, transmitted to the cochlear duct. These hair cells provide stimulation of auditory primary axons that transmit sound.

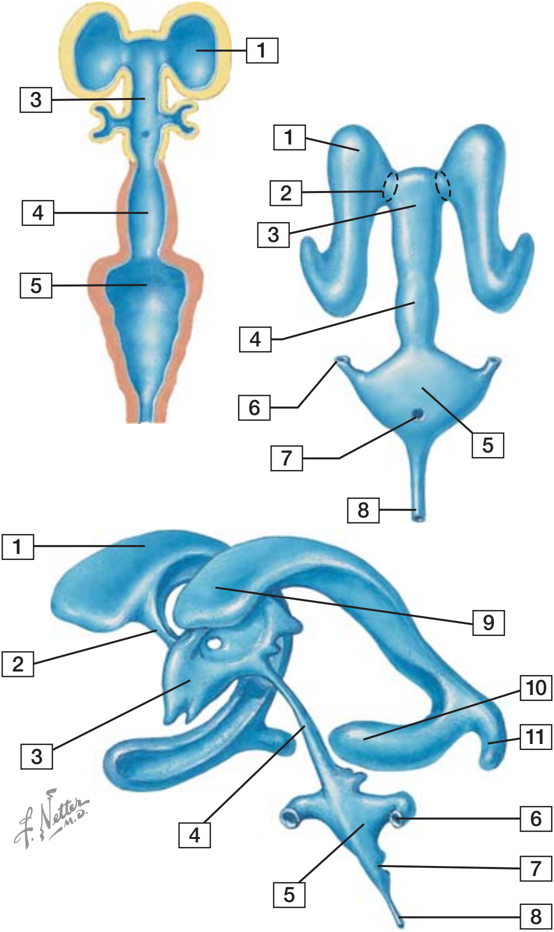
Development of the Pituitary Gland



Development of the Pituitary Gland

1. Infundibular process
2. Brain
3. Rathke's pouch
4. Infundibulum
5. Pars distalis
6. Pars tuberalis
7. Pars nervosa
8. Pars intermedia

Comment: The posterior lobe of the pituitary (neurohypophysis, pars nervosa) derives from the brain and contains axonal processes from the hypothalamus that secrete oxytocin and vasopressin into the general circulation. The anterior lobe (adenohypophysis) derives from the roof of the stomodeum and encircles the base of the neurohypophysis. The adenohypophysis contains pituicytes that respond to releasing and inhibitory factors produced by neurons, secreted into the hypophyseal-portal vascular system (a private vascular channel) at the contact zone of the median eminence. Pituicytes can be the source of tumor development (e.g., growth hormone–secreting cells), producing endocrine deficits and mass effects from impingment on the nearby optic chiasm.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Development of the Ventricles

1. Lateral ventricle
2. Interventricular foramen of Monro
3. Third ventricle
4. Cerebral aqueduct
5. Fourth ventricle
6. Lateral aperture (foramen of Luschka) of the fourth ventricle
7. Median aperture (foramen of Magendie) of the fourth ventricle
8. Central canal of the spinal cord
9. Anterior horn of left lateral ventricle in frontal lobe
10. Inferior horn of left lateral ventricle in temporal lobe
11. Posterior horn of left lateral ventricle in occipital lobe

Comment: From the original central canal of the neural tube, the ventricular system develops in response to neural growth, development, folding, and expansion of the forebrain. Cerebrospinal fluid (CSF) is produced internally, channeled through the ventricular system from rostral to caudal into the subarachnoid space, and absorbed into the venous circulation via the arachnoid granulations acting as one-way valves into the venous sinuses. There are a few sites of vulnerability where CSF flow can be disrupted. Atresia of the cerebral aqueduct leads to internal hydrocephalus (increased intracranial pressure with expansion of the third ventricle and lateral ventricles). Obstruction of outflow of CSF from the foramina of Luschka and of Magendie of the fourth ventricle (Dandy-Walker syndrome) leads to hydrocephalus affecting the entire ventricular system. Obstruction of CSF absorption into the venous sinuses (e.g., acute purulent meningitis) can lead to external hydrocephalus.

2

Regional Neuroscience

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Regional Neuroscience
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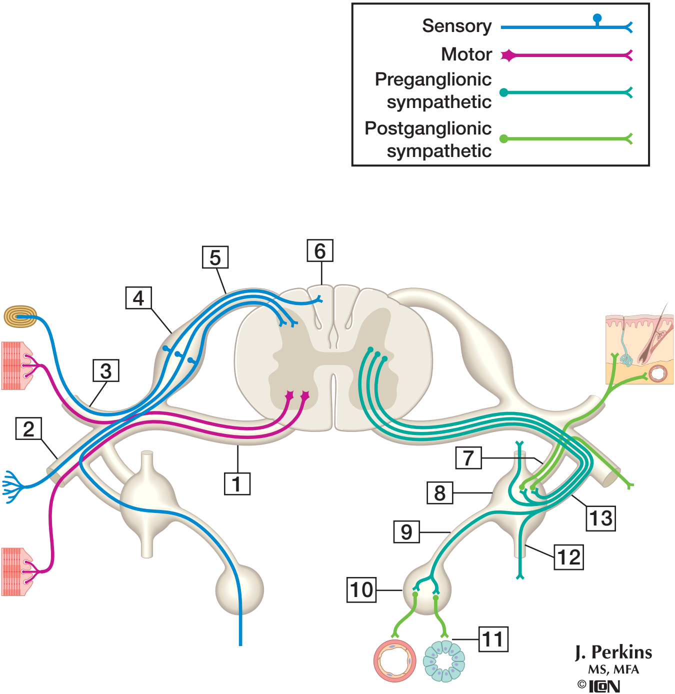
Spinal Cord and PNS Schematic



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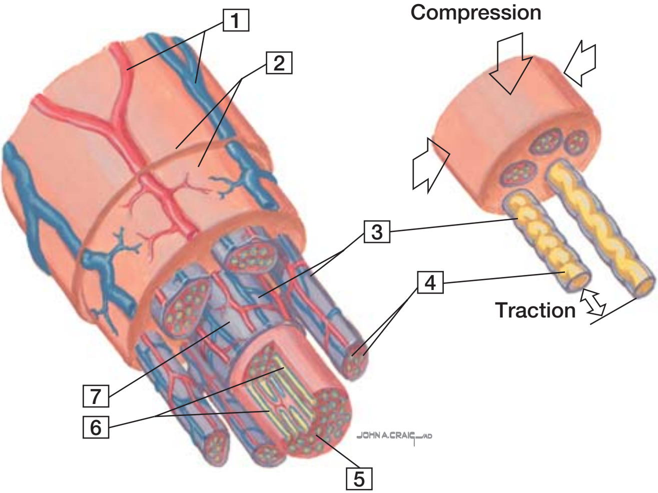
Spinal Cord and PNS Schematic

1. Ventral root
2. Ventral ramus
3. Dorsal ramus
4. Dorsal root ganglion
5. Dorsal root
6. Dorsal column
7. Gray ramus communicans
8. Sympathetic chain ganglion
9. Splanchnic nerve
10. Collateral sympathetic ganglion
11. Neuroeffector junctions
12. Sympathetic chain
13. White ramus communicans

Comment: Peripheral nerves form from the union of dorsal and ventral roots, bringing together both afferent and efferent peripheral nerve fibers. Subsequent branching carries the nerve fibers to their appropriate targets. Peripheral nerves contain lower motor neuron axons (alpha to skeletal muscle fibers, gamma to muscle spindle contractile fibers); autonomic axons (preganglionic and postganglionic); and primary sensory axons (myelinated and unmyelinated).

Peripheral nerves may be damaged by metabolic or chemical insult (diabetic neuropathy, alcoholic neuropathy), often manifested as distal symmetrical peripheral neuropathies. Some insults may damage only unmyelinated axons (leprosy) or myelinated axons (demyelinating neuropathy such as Guillain-Barré syndrome). Physical damage or destructive lesions affect functions in the territory of distribution and may result in flaccid paralysis of skeletal muscles, autonomic dysfunction, or loss of some or all aspects of somatic sensation. Irritative lesions of peripheral nerves often cause pain that radiates into the territory of innervation (sciatica).

Anatomy of a Peripheral Nerve

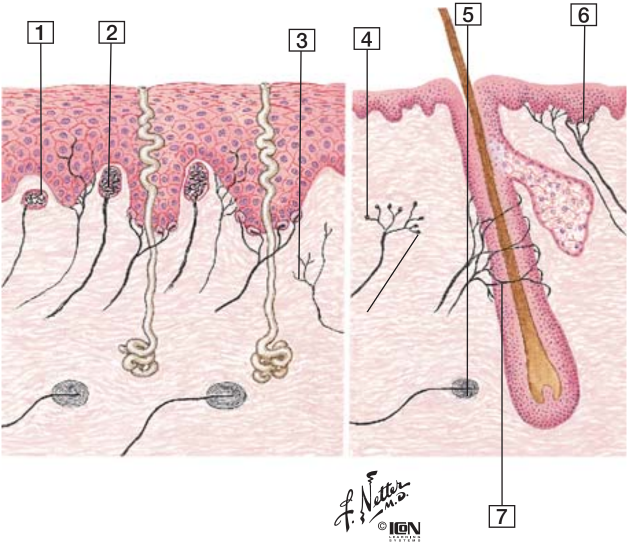


Epineurial coat provides some protection against compression. Spiral configuration of nerve fiber bundles within fascicles provides some protection from traction.

Anatomy of a Peripheral Nerve

1. Longitudinal vessels
2. Outer and inner layers of epineurium
3. Fascicles
4. Nerve fiber bundles
5. Endoneurium
6. Individual axons
7. Perineurium

Comment: The epineurium is a tough outer layer of supportive connective tissue that enwraps the entire nerve. The perineurium enwraps fascicles of multiple axons and acts as a blood-nerve barrier; disruption of this barrier, sometimes seen in diabetic neuropathies and other neuropathies, exposes the axons to diffusion of potentially damaging substances. The endoneurium is loose supportive connective tissue found among individual axons within a fascicle. The vasa nervorum (longitudinal vessels) supply blood to the nerve and are susceptible to pressure and other insults. An entrapment syndrome, such as carpel tunnel syndrome, can compromise blood flow to the nerve (median) and cause ischemic damage, including severe pain.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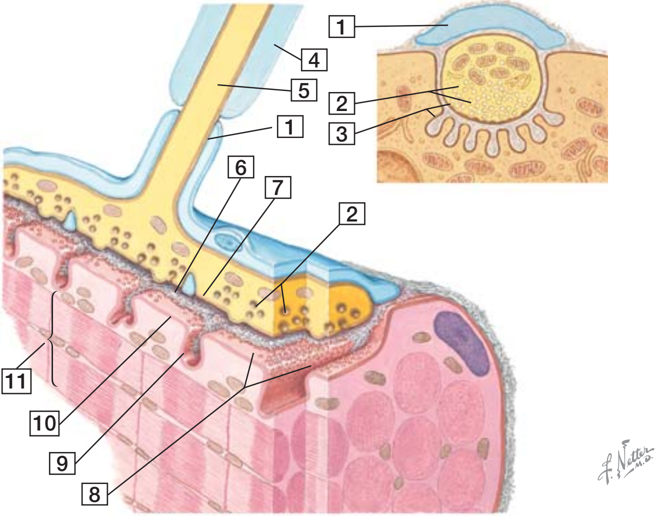
Cutaneous Receptors

1. Krause’s end bulbs
2. Meissner’s corpuscle
3. Free nerve ending
4. Ruffini ending
5. Pacinian corpuscle
6. Merkel’s disc
7. Nerve plexus around hair follicle

Comment: Cutaneous receptors are specialized sense organs found at the distal end of primary sensory axons. They act as dendrites; a threshold stimulus leads to firing of an action potential at the initial segment of the axon. For many decades, specific types of sensory receptors were associated with specific types of conscious somatic sensations, but this has not been borne out with full accuracy. The best correlations indicate that encapsulated receptors such as Pacinian corpuscles are fast-adapting mechanoreceptors that detect vibratory sensation or brief touch. Merkel’s discs are slowly adapting mechanoreceptors that detect maintained deformation or sustained touch. Meissner’s corpuscles are fast-adapting mechanoreceptors that detect moving touch. Ruffini endings are slowly adapting mechanoreceptors that detect steady pressure applied to hairy skin. Hair follicle receptors are rapidly adapting mechanoreceptors that detect hair movement. Krause’s end bulbs were thought to be thermoreceptors, although that has been questioned. Bare nerve endings can detect thermal sensation (especially extremes) and nociceptive stimuli.

Neuromuscular Junction

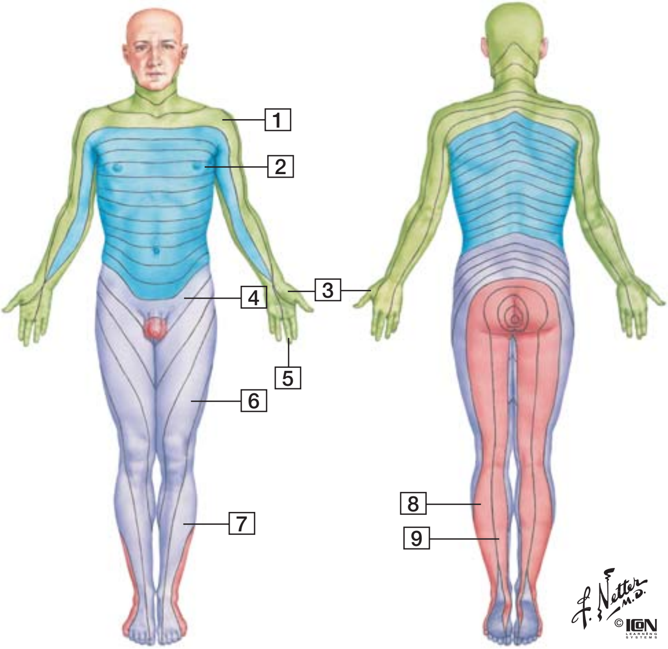


Neuromuscular Junction

1. Schwann cell
2. Synaptic vesicles
3. Synaptic cleft
4. Myelin sheath
5. Axoplasm
6. Basement membrane
7. Presynaptic membrane
8. Acetylcholine receptor sites
9. Junctional folds (secondary folds)
10. Postsynaptic membrane
11. Myofibrils

Comment: When motor action potential invades the motor end plate, the influx of Ca^{++} causes the simultaneous release of approximately 200 vesicles of acetylcholine (Ach). The vesicles fuse with the nerve terminal membrane and release Ach into the synaptic cleft, especially into the secondary folds which are lined with nicotinic cholinergic receptors. The quantal release of Ach is usually sufficient to bring the muscle membrane potential to threshold, producing a muscle action potential that results in contraction of the skeletal muscle fiber. A single muscle fiber has only one neuromuscular junction, but one motor axon may innervate from a few (extraocular muscles) to a few thousand (quadriceps) skeletal muscle fibers. The motor end plate also exerts a trophic influence on its innervated skeletal muscle fibers; loss of innervation leads to muscle atrophy, loss of contraction, and total inactivity. If a lower motor neuron dies and retracts its motor endplates from its innervated muscle fibers (the motor unit), axons from adjacent intact lower motor neurons may sprout and reinnervate those originally denervated sites. This commonly happens in polio, resulting in partial paralysis (flaccid). The reinnervation leaves a motor neuron with a larger-sized motor unit.

Dermatomal Distribution

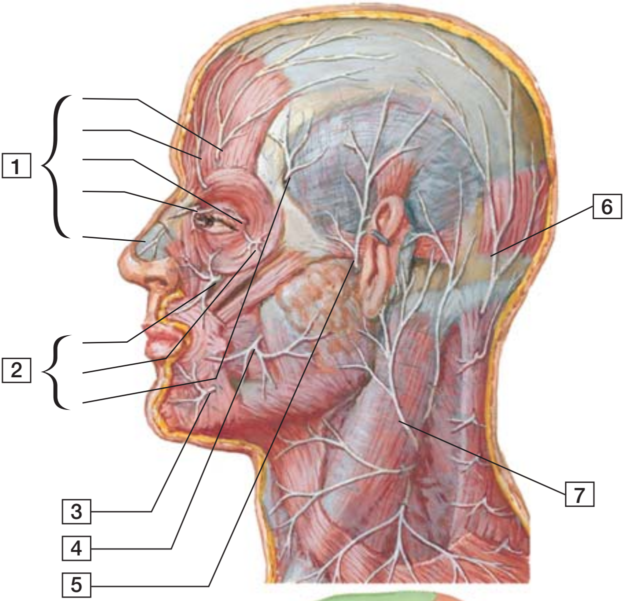


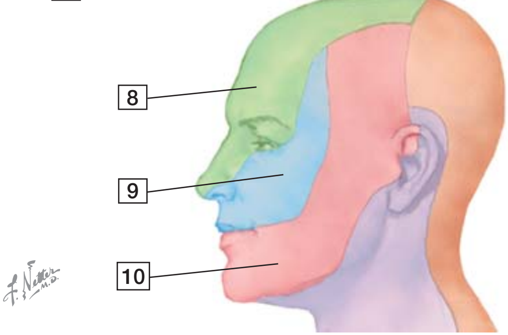
Dermatomal Distribution

1. C5
2. T4
3. C6
4. L1
5. C7
6. L4
7. L5
8. S1
9. S2

Comment: Dermatomes are regions of the body to which specific nerve roots predominantly distribute their primary afferent fibers; they are based on the corresponding spinal cord segments that receive the primary sensory information through the dorsal roots. In the spinal cord, sensory fibers from a specific dermatomal region actually distribute to at least three segments of the spinal cord, including the predominant segment, and a segment above and below. Thus, a destructive lesion of one nerve root does not leave the dermatome totally devoid of sensation; that phenomenon would require destruction of the specific associated nerve root, as well as the nerve roots above and below that dermatome. However, an irritative lesion, such as the impingement of a herniated disc (nucleus pulposus) on a dorsal root, will produce sharp, radiating pain in the distribution of the principal dermatome. The nonuniform distribution of dermatomes across the body reflects the rotation of the limb buds during development. Knowledge of the dermatomes is important for distinguishing between nerve root lesions (which may individually produce diminished sensation in a region of the body) and a peripheral nerve lesion (which may produce total anesthesia in the distribution of that nerve).

Cutaneous Nerves of the Head and Neck



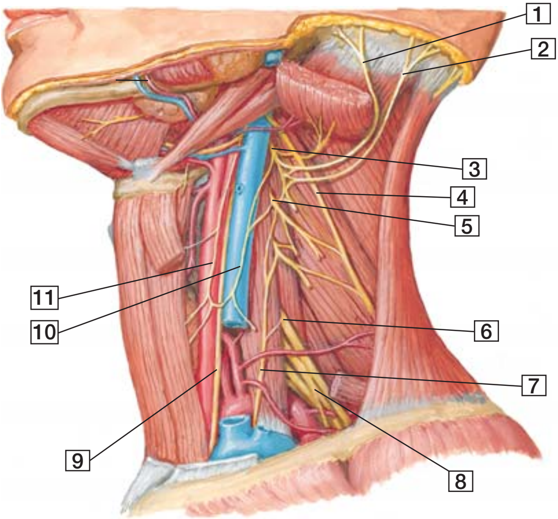


Cutaneous Nerves of the Head and Neck

1. Ophthalmic division of V
2. Maxillary division of V
3. Mandibular division of V—mental nerve
4. Mandibular division of V—buccal nerve
5. Mandibular division of V—auriculotemporal nerve
6. Greater occipital nerve
7. Greater auricular nerve
8. Ophthalmic nerve (V1)
9. Maxillary nerve (V2)
10. Mandibular nerve (V3)

Comment: The three divisions of the trigeminal nerve (CN V), the ophthalmic, maxillary, and mandibular nerves, distribute exclusively to their respective territories of innervation without any overlap in sensory distribution. Thus, a lesion in one of the divisions produces total anesthesia in the distribution of that nerve, as occurs with a mandibular block for dental procedures. This contrasts with the spinal dermatomes, which have at least three segments of overlap. Similar to spinal nerve roots, the three divisions of the trigeminal nerve may be subject to irritative lesions, as sometimes occurs with trigeminal neuralgia (tic douloureux), a condition in which even mild stimulation of a branch of the trigeminal nerve evokes excruciating lancinating pain in the distribution of that nerve. This incapacitating pain sometimes occurs because of pressure on the affected nerve and may be alleviated by a decompressive surgical procedure, as well as by medical treatment with pharmaceutical agents that diminish nerve firing.

Cervical Plexus



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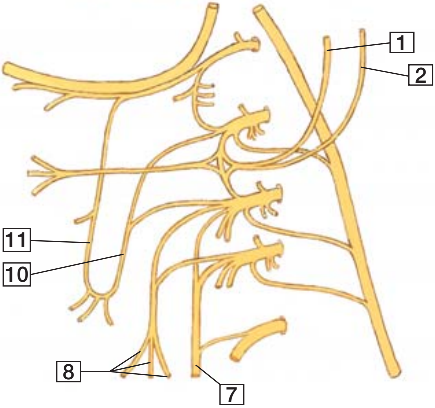
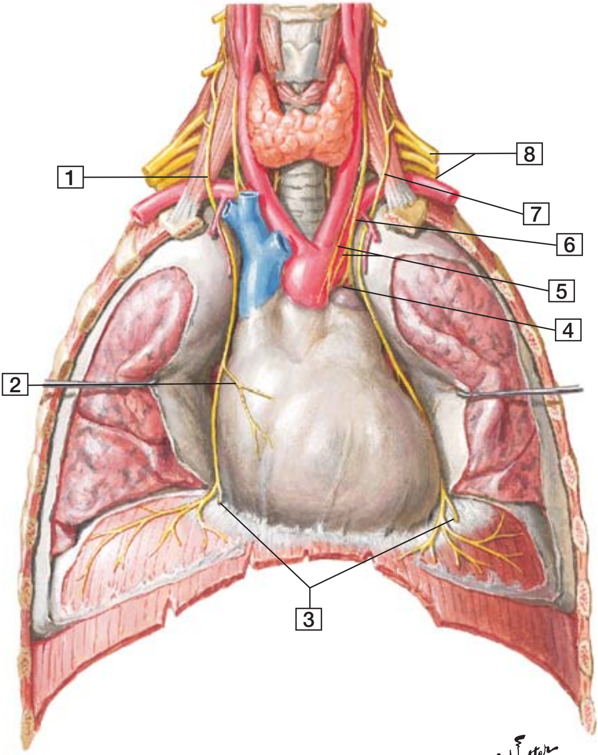


Cervical Plexus

1. Greater auricular nerve
2. Lesser occipital nerve
3. C2 spinal nerve (ventral ramus)
4. Accessory nerve
5. C3 spinal nerve (ventral ramus)
6. C5 spinal nerve (ventral ramus)
7. Phrenic nerve
8. Supraclavicular nerves
9. Vagus nerve
10. Inferior root, ansa cervicalis
11. Superior root, ansa cervicalis

Comment: The cervical plexus consists of the ventral (anterior) primary rami of the C1-C4 nerves. Sensory branches include the greater and lesser occipital nerves, the greater auricular nerve, cutaneous cervical nerves, and supraclavicular nerves. Motor branches include the ansa hypoglossi, branches to the scalenus medius and levator scapulae muscles, the phrenic nerve, and branches to the spinal accessory nerve. The cervical plexus lies deep to the sternocleidomastoid muscle, and lesions are infrequent, the result of trauma or mass lesions or a sequela of surgery. Involvement of sensory branches may cause loss of cutaneous sensation (ablative lesion) or pain and paresthesias (irritation) in regions of the head and neck supplied by these branches. Involvement of motor branches may result in muscle dysfunction such as shoulder elevation, as well as head rotation and flexion (spinal accessory nerve damage).



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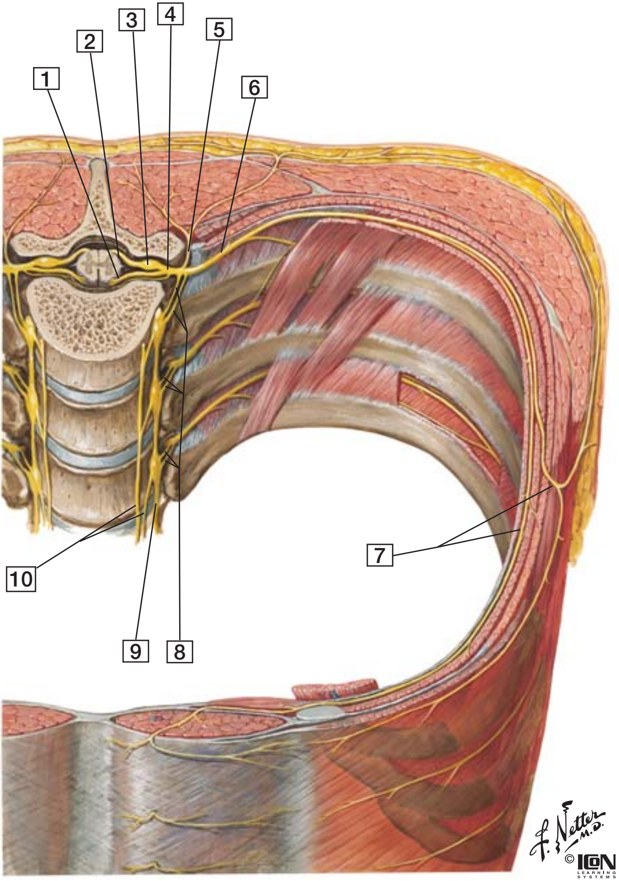
Phrenic Nerve

1. Right phrenic nerve
2. Pericardial branch of phrenic nerve
3. Phrenicoabdominal branches of phrenic nerves (to inferior surface of diaphragm)
4. Left recurrent laryngeal nerve
5. Thoracic cardiac nerves
6. Left vagus nerve (CN X)
7. Left phrenic nerve
8. Brachial plexus

Comment: The phrenic nerves, formed from the C3–C5 ventral roots, provide the motor nerve supply to the diaphragm. Damage to the phrenic nerves usually occurs in the mediastinum, not the cervical plexus; major causes of damage include enlarged mediastinal nodes, an aortic aneurysm, mediastinal tumors, sequelae of surgery, or demyelination from Guillain-Barré syndrome. Unilateral damage, damaging the diaphragm on one side (ipsilateral), can usually be tolerated at rest but not following exertion. Bilateral phrenic nerve damage leads to diaphragmatic paralysis with extreme dyspnea and hypoventilation.

See book 9.19 Upper figure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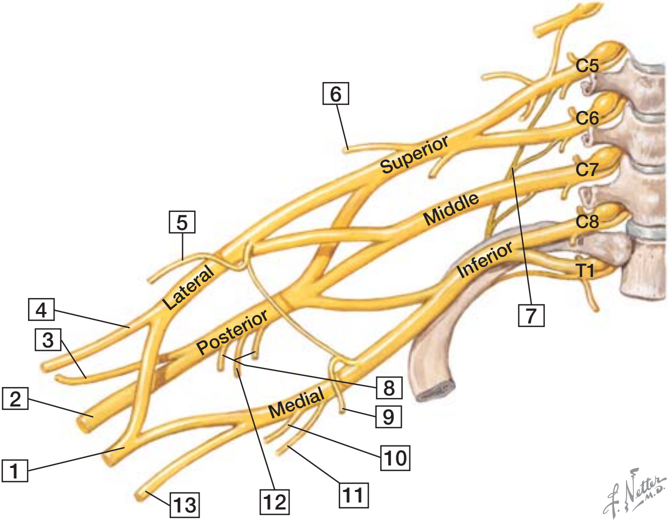
Thoracic Nerves

1. Ventral root
2. Dorsal root
3. Spinal ganglion (dorsal root)
4. Spinal nerve trunk
5. Dorsal ramus of spinal nerve
6. Intercostal nerve (ventral ramus)
7. Lateral cutaneous branch and anterior cutaneous branch of intercostal nerves
8. Rami communicantes (gray and white)
9. Sympathetic trunk
10. Thoracic splanchnic nerves

Comment: Nerves derived from the thoracic segments provide intercostal distribution. Sensory branches supply the thoracic dermatomes, deeper muscular structures, periosteum, parietal pleura, the peritoneum, blood vessels, and breast tissue. Muscular branches distribute to muscles of the thoracic and abdominal walls. When the phrenic nerve is damaged, or during times of dyspnea, these muscles can act as accessory respiratory muscles and assist in breathing.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C5
C6
Superior
C7
Middle
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C8
Inferior
T1
Lateral
4
3
Posterior
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Medial
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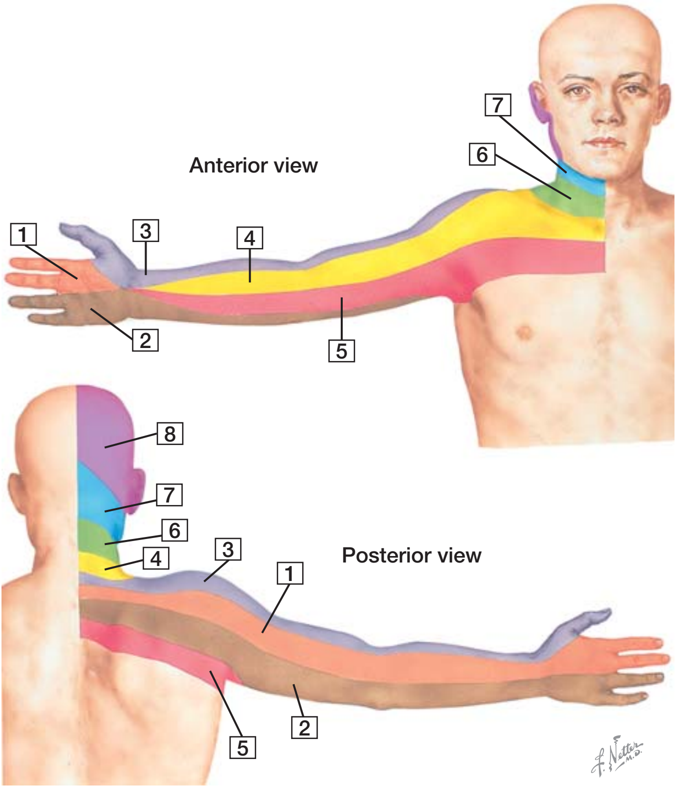
Brachial Plexus

1. Median nerve
2. Radial nerve
3. Axillary nerve
4. Musculocutaneous nerve
5. Lateral pectoral nerve
6. Suprascapular nerve
7. Long thoracic nerve
8. Subscapular nerves
9. Medial pectoral nerve
10. Medial cutaneous nerve of the forearm
11. Medial cutaneous nerve of the arm
12. Thoracodorsal nerve
13. Ulnar nerve

Comment: The brachial plexus is formed from the ventral roots of C5–C8 and T1, with a small contribution from C4. Sensory and sympathetic nerve fibers also distribute with the plexus. The roots of the plexus give rise to three trunks, three ventral and three dorsal divisions, three cords, and numerous terminal branches, the peripheral nerves. Lesions of the upper brachial plexus (affecting C5 and C6 contributions) may lead to paresis of shoulder abduction and external rotation, as well as paresis of elbow flexion, due to impaired motor supply to the deltoid, supraspinatus, infraspinatus, biceps, supinator, and brachioradialis muscles. The arm hangs down and is rotated medially and the forearm is pronated. Lesions of the lower brachial plexus (affecting C8 and T1 contributions) may result in paralysis of finger flexion and paralysis of all small muscles of the hand, producing a claw hand.

Dermatomes of the Upper Limb



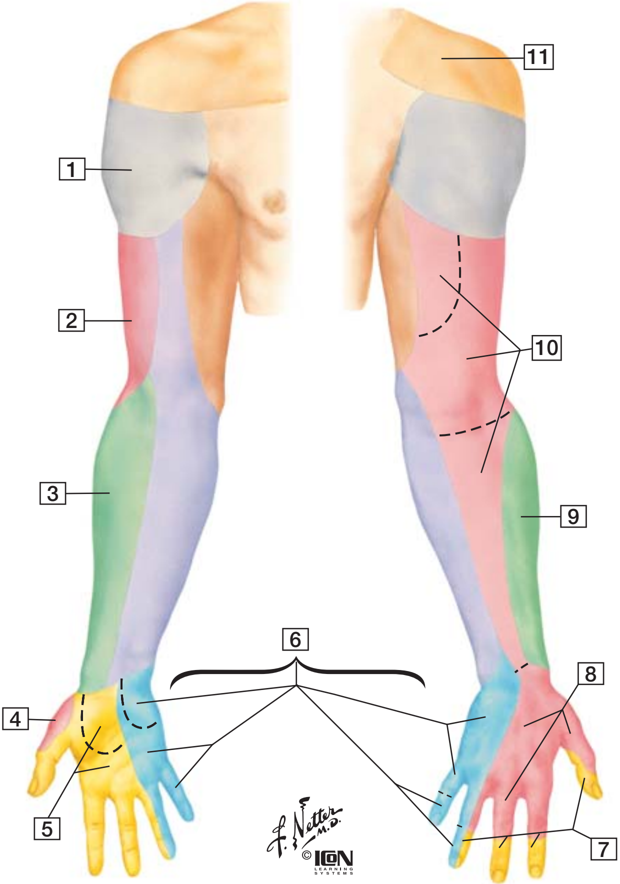
Dermatomes of the Upper Limb

1. C7
2. C8
3. C6
4. C5
5. T1
6. C4
7. C3
8. C2

Comment: The dermatomes of the upper extremity are arranged on the basis of limb bud extension embryologically, resulting in disruption of orderly segmental distribution of the cervical dermatomes. Thus, C5 and T1 dermatomes are adjacent to each other. Because of extensive sensory overlap among dermatomes (at least three segments), damage to one nerve root does not produce total loss of sensation in the distribution of that dermatome. However, an irritative lesion of a single nerve root may result in radiating pain in the distribution of that dermatome.

See book 9.22

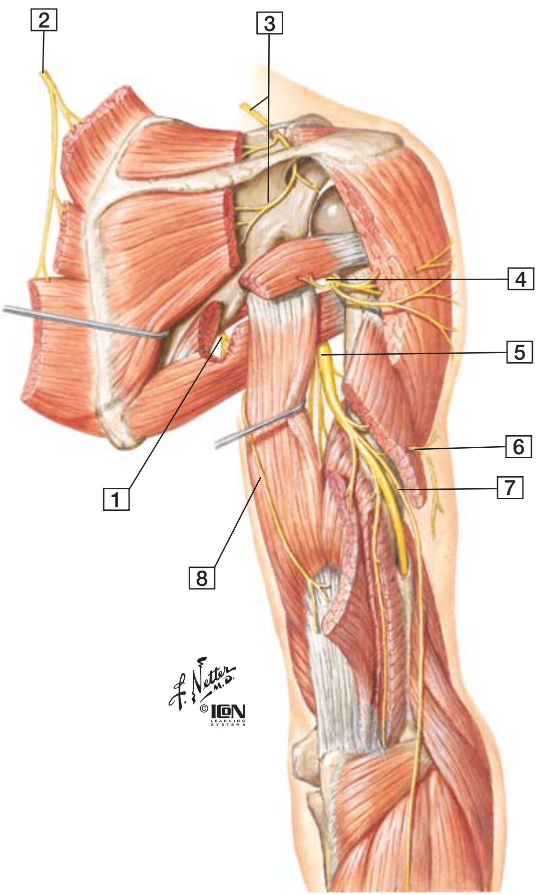
Cutaneous Innervation of the Upper Limb from Peripheral Nerves



Cutaneous Innervation of the Upper Limb from Peripheral Nerves

1. Axillary nerve—superior lateral cutaneous nerve of the arm
2. Radial nerve—inferior lateral cutaneous nerve of the arm
3. Lateral cutaneous nerve of the forearm
4. Radial nerve
5. Median nerve
6. Ulnar nerve
7. Median nerve
8. Radial nerve
9. Lateral cutaneous nerve of the forearm
10. Radial nerve—posterior cutaneous nerve of the arm; inferior lateral cutaneous nerve of the arm; posterior cutaneous nerve of the forearm
11. Supraclavicular nerves

Comment: The cutaneous innervation of the upper limb is provided by the musculocutaneous, axillary, radial, median, and ulnar nerves, terminal branches of the brachial plexus. Unlike the overlapping territories of the dorsal nerve roots, these terminal peripheral nerves supply specific regions of the upper limb that do not overlap. Thus, a peripheral nerve injury or lesions result in total anesthesia in the territory of that peripheral nerve. An irritative lesion (e.g., carpal tunnel syndrome affecting the median nerve) may result in pain and paresthesias in the territory of that peripheral nerve.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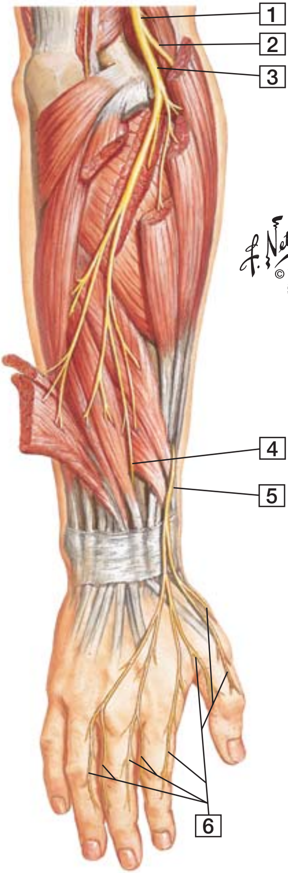
Scapular, Axillary, and Radial Nerves above the Elbow

1. Lower subscapular nerve
2. Dorsal scapular nerve
3. Suprascapular nerve
4. Axillary nerve
5. Radial nerve
6. Lower lateral cutaneous nerve of the arm
7. Posterior cutaneous nerve of the forearm
8. Posterior cutaneous nerve of the arm (branch of radial nerve)

Comment: The axillary nerve (C5–C6) supplies the deltoid and teres minor muscles; it aids in abduction of the arm to the horizontal, and in outward rotation of the arm. Axillary nerve damage may result in deltoid atrophy and weakness in abduction from 15 to 90 degrees and loss of cutaneous sensation over the lower half of the deltoid. The radial nerve (C5–T1) supplies the triceps, anconeus, brachioradialis, extensor carpi radialis, extensor digitorum, and supinator muscles; it aids in extension and flexion of the elbow. A radial nerve lesion may lead to paralysis of extension and flexion of the elbow, as well as paralysis of supination of the forearm. The wrist and fingers cannot be extended, and a wristdrop occurs.

See book 9.24

Radial Nerve in the Forearm



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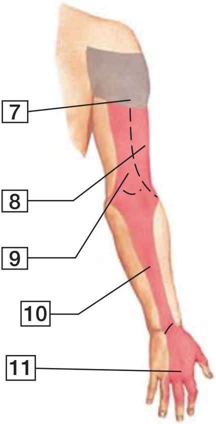
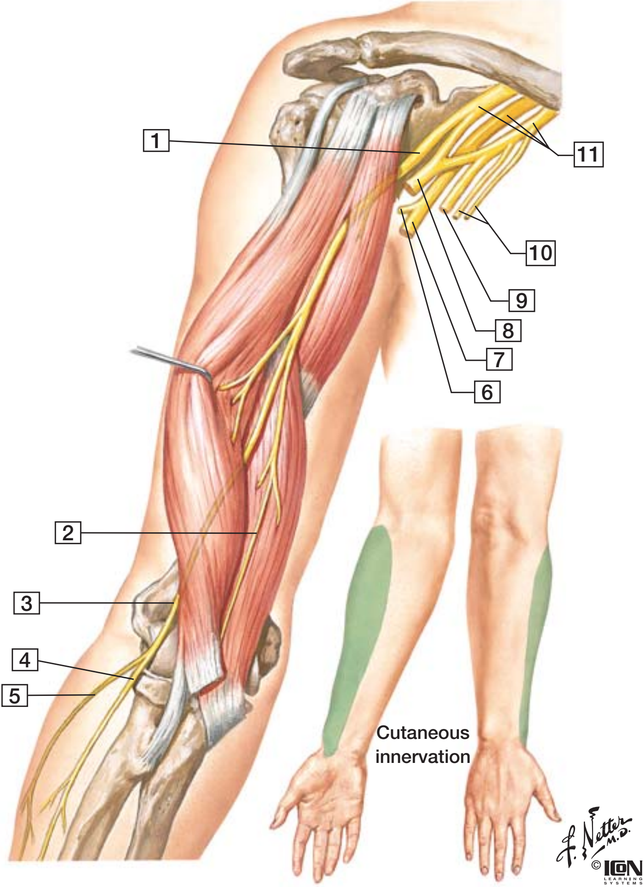


Radial Nerve in the Forearm

1. Radial nerve
2. Superficial branch
3. Deep terminal branch
4. Posterior interosseous nerve (deep branch)
5. Superficial branch of radial nerve
6. Dorsal digital nerves
7. Upper lateral cutaneous nerve of the arm
8. Lower lateral cutaneous nerve of the arm
9. Posterior cutaneous nerve of the arm
10. Posterior cutaneous nerve of the forearm
11. Superficial branch of the radial nerve

Comment: The radial nerve (C5–T1, mainly C6–C8) supplies motor innervation to flexors and extensors of the elbow, supinators of the forearm, extensors of the wrist and fingers, and abductors of the thumb; a lesion leads to impairment of these functions. The radial nerve supplies cutaneous sensation to the posterior upper arm, an elongated region of the posterior forearm, and the posterior hand, thumb, and lateral 2½ fingers; a lesion may lead to loss of sensation (or pain and paresthesias) within these areas.

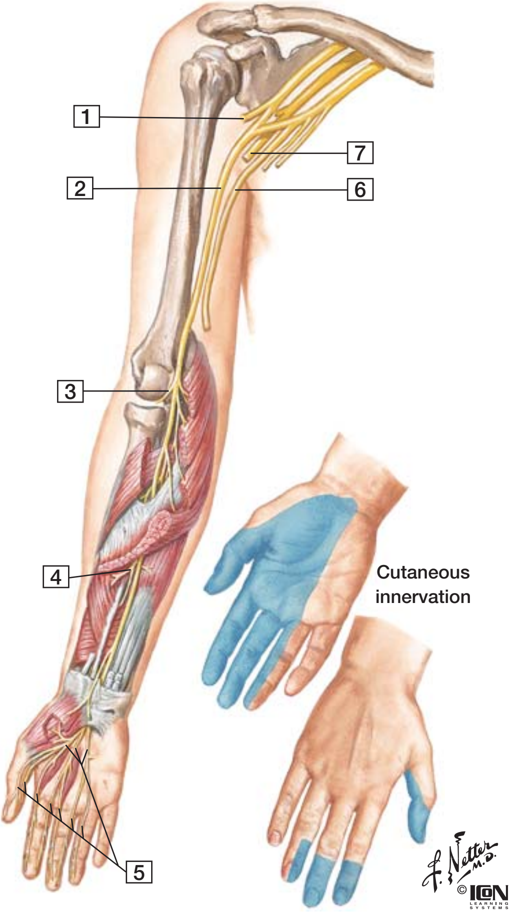
Musculocutaneous Nerve



Musculocutaneous Nerve

1. Musculocutaneous nerve
2. Articular branch
3. Lateral cutaneous nerve of the forearm
4. Anterior branch
5. Posterior branch
6. Axillary nerve
7. Radial nerve
8. Median nerve
9. Ulnar nerve
10. Medial cutaneous nerves of the forearm and arm
11. Medial, posterior, and lateral cords of the brachial plexus

Comment: The musculocutaneous nerve (C5–C6) supplies motor innervation to the biceps brachii, coracobrachialis, and brachialis muscles; it aids in flexion of the upper and lower arm, supination of the lower arm, and elevation and abduction of the arm. A lesion of the musculocutaneous nerve leads to atrophy of these muscles and weakness of flexion in the supinated arm. Loss of sensation on the lateral forearm also may occur.



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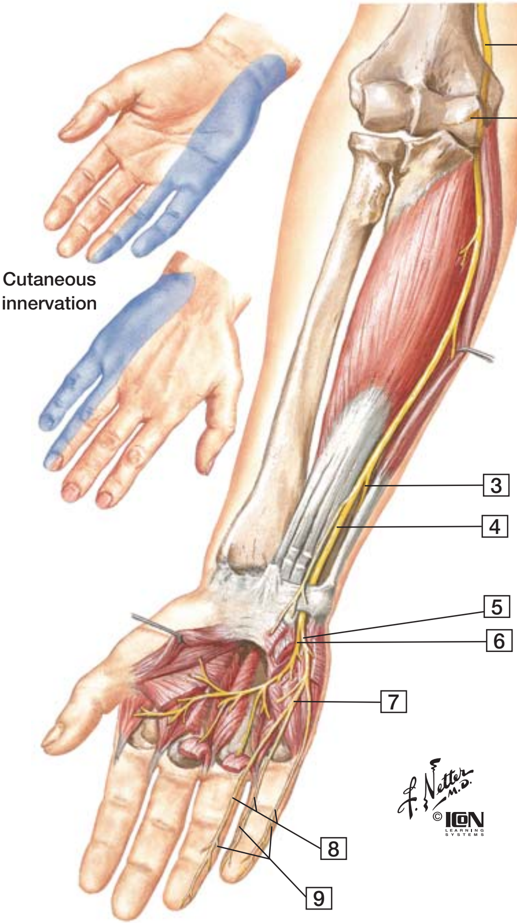
Median Nerve

1. Musculocutaneous nerve
2. Median nerve
3. Articular branch
4. Anterior interosseous nerve
5. Palmar digital nerves (common and proper)
6. Ulnar nerve
7. Radial nerve

Comment: The median nerve supplies motor innervation to flexors of the fingers, abductors and opponens muscles of the thumb, and pronators of the forearm. The median nerve supplies sensory innervation to the palmar surface of the hand (thumb, index finger, middle finger, and half of the fourth finger). The median nerve is vulnerable to trapping by the flexor retinaculum in the forearm (carpal tunnel syndrome), resulting in weakness of flexion of the fingers; weakness in abducting and opposition of the thumb; and wasting of the thenar muscles. Carpal tunnel syndrome results in pain and paresthesias (or sometimes loss of sensation) in the radial distribution of the hand. The pain sometimes radiates back to the wrist.

Ulnar Nerve

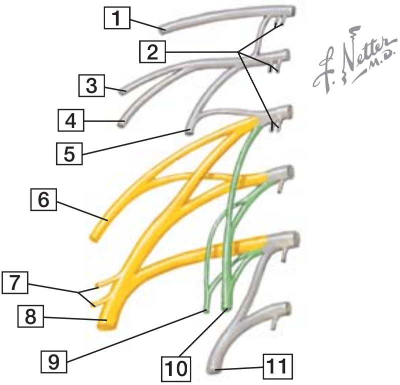


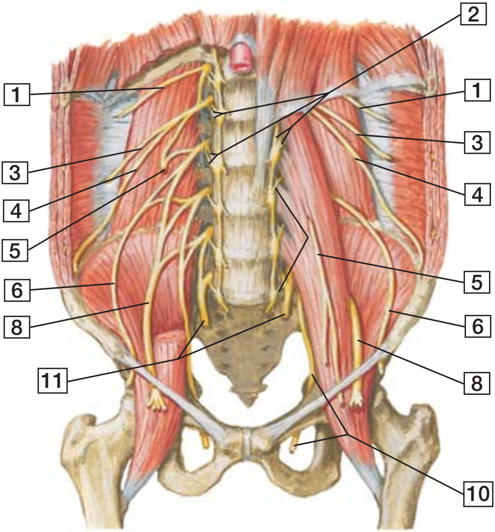
Ulnar Nerve

1. Ulnar nerve
2. Articular branch
3. Dorsal branch
4. Palmar branch
5. Superficial branch
6. Deep branch
7. Common palmar digital nerve
8. Proper palmar digital nerves
9. Branches to dorsum of middle and distal phalanges

Comment: The ulnar nerve supplies motor innervation to muscles of the hand, wrist flexors, and abductors and adductors of the fingers. The ulnar nerve supplies sensory fibers to the dorsal and palmar regions of the hand and the medial fourth finger and the little finger. A lesion of the ulnar nerve results in wasting of hand muscles, weakness of wrist flexion and ulnar deviation of the hand, and weakness of abduction and adduction of the fingers (hyperextension of the fingers at metacarpophalangeal joints and flexion at the interphalangeal joints). This positioning of the hand is known as “claw hand.” A lesion also results in loss of sensation (or pain and paresthesias) in the sensory distribution of the ulnar nerve.

Lumbar Plexus

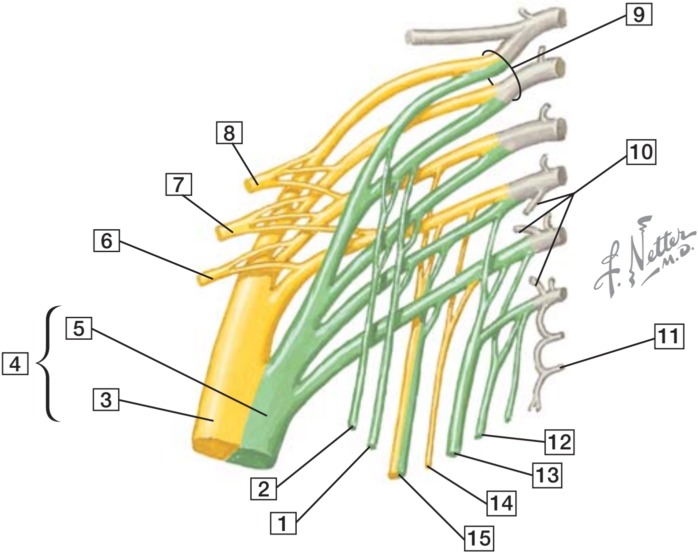




Lumbar Plexus

1. Subcostal nerve
2. White and gray rami communicantes
3. Iliohypogastric nerve
4. Ilioinguinal nerve
5. Genitofemoral nerve
6. Lateral cutaneous nerve of the thigh
7. Muscular branches to psoas and iliacus muscles
8. Femoral nerve
9. Accessory obturator nerve
10. Obturator nerve
11. Lumbosacral trunks

Comment: The lumbar plexus is formed from the ventral (anterior) primary rami of L1–L4 nerve roots. The L1 and L2 roots form the iliohypogastric, ilioinguinal, and genitofemoral nerves. The L2–L4 roots form the femoral, obturator, and lateral femoral cutaneous nerves. Lumbar plexus lesions are rare because of the protection provided by the psoas muscle. The most characteristic motor loss of such a lesion involves weakness of hip flexion and adduction and weakness of leg extension. Some sensory loss may be seen over the anterior and medial aspect of the thigh. Lumbar plexus lesions must be distinguished from nerve root lesions, which are usually painful and follow a nerve root distribution.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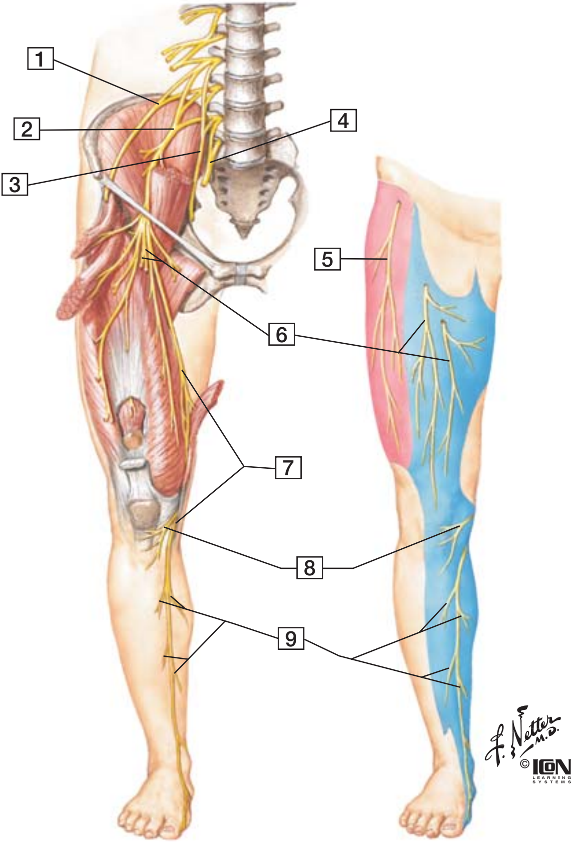
Sacral and Coccygeal Plexuses

1. Nerve to obturator internus
2. Nerve to quadratus femoris
3. Common fibular (peroneal) nerve
4. Sciatic nerve
5. Tibial nerve
6. Nerve to piriformis
7. Inferior gluteal nerve
8. Superior gluteal nerve
9. Lumbosacral trunk
10. Pelvic splanchnic nerves (parasympathetic)
11. Coccygeal nerve
12. Nerve to levator ani and (ischio-)coccygeus muscles
13. Pudendal nerve
14. Perforating cutaneous nerve
15. Posterior cutaneous nerve of the thigh

Comment: The sacral and coccygeal plexuses are formed from the roots of the L4–S4 segments. Major branches include the superior (L4–S1) and inferior (L5–S2) gluteal nerves, the posterior femoral cutaneous nerve (S1–S3), the sciatic nerve (L4–S3) and its tibial and common peroneal divisions, and the pudendal nerve (S2–S4). Lesions of the sacral plexus usually present as weakness in the posterior thigh and in the muscles of the legs and feet (distribution of the gluteal, tibial, and peroneal nerves). The leg weakness may be profound, including weakness of hip extension and abduction, weakness of leg flexion, and weakness of ankle plantarflexion and dorsiflexion. More proximal lesions may result in weakness of gluteal muscles. Sensory loss may occur in the posterior thigh, anterolateral and posterior leg, the plantar and dorsolateral surface of the foot, and saddle/perianal region.

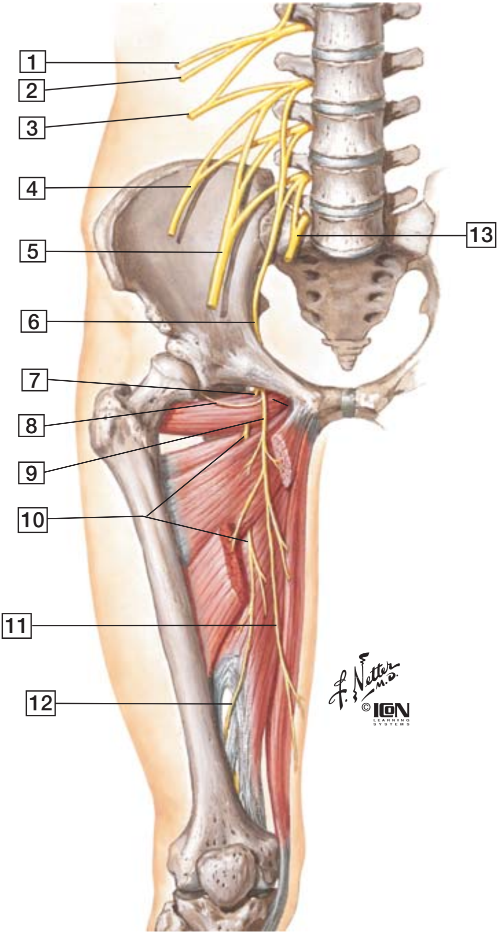
Femoral and Lateral Femoral Cutaneous Nerves



Femoral and Lateral Femoral Cutaneous Nerves

1. Lateral femoral cutaneous nerve
2. Femoral nerve
3. Obturator nerve
4. Lumbosacral trunk
5. Lateral femoral cutaneous nerve
6. Anterior cutaneous branches of the femoral nerve
7. Saphenous nerve
8. Infrapatellar branch of the saphenous nerve
9. Medial crural cutaneous branches of the saphenous nerve

Comment: The femoral nerve (L2–L4) provides motor innervation to the iliopsoas, sartorius, and quadriceps femoris; it aids in flexion and outward rotation of the hip, flexion and inward rotation of the lower leg, and extension of the lower leg around the knee joint. A femoral nerve lesion results in weakness of extension of the leg, weakness of flexion of the hip and leg, and atrophy of the quadriceps. A femoral nerve lesion also results in loss of cutaneous sensation on the anterior thigh and the anterior and medial surfaces of the leg and foot. The lateral femoral cutaneous nerve provides sensation to the anterior and lateral surfaces of the thigh down to the knees. Compression of this nerve at the inguinal ligament or near the surface (e.g., by a tight belt) may result in pain and paresthesias (or loss of sensation) on the anterior and lateral surfaces of the ipsilateral thigh.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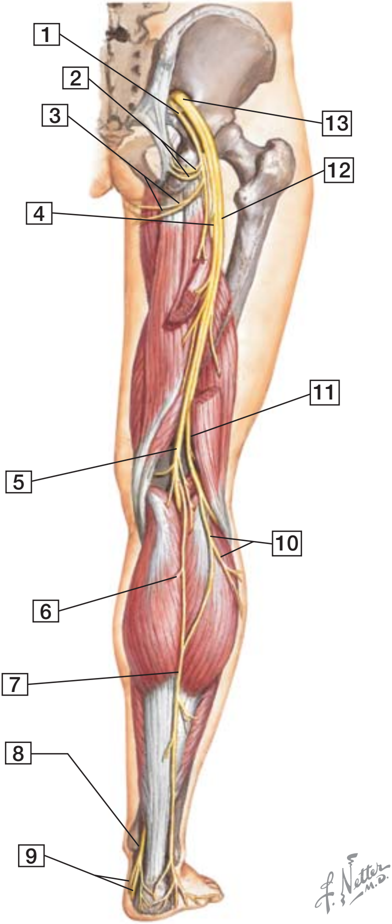
Obturator Nerve

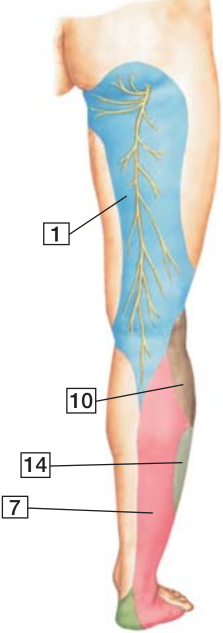
1. Iliohypogastric nerve
2. Ilioinguinal nerve
3. Genitofemoral nerve
4. Lateral femoral cutaneous nerve
5. Femoral nerve
6. Obturator nerve
7. Posterior branch
8. Articular branch
9. Anterior branch
10. Posterior branch
11. Cutaneous branch
12. Articular branch to the knee joint
13. Lumbosacral trunk

Comment: The obturator nerve (L2–L4) supplies the pectineus, adductor muscles of the thigh, gracilis, and the external obturator muscles; it aids in adduction and rotation of the thigh. A lesion of the obturator produces weakness of adduction of the thigh and a tendency toward thigh abduction with walking, as well as weakness of external rotation of the thigh. A small zone of skin on the medial thigh may show a loss of sensation.

Sciatic and Posterior Femoral Cutaneous Nerves



Cutaneous innervation



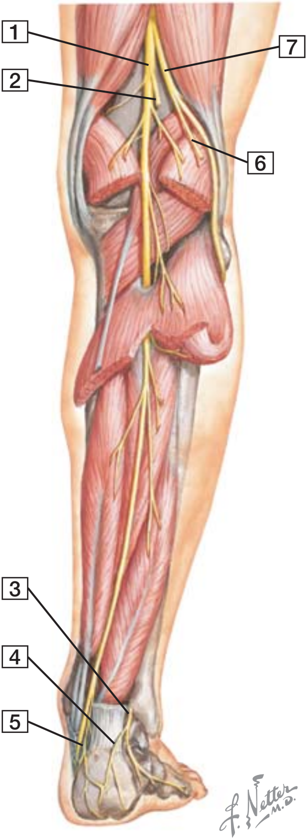
Sciatic and Posterior Femoral Cutaneous Nerves

1. Posterior femoral cutaneous nerve
2. Inferior cluneal nerve
3. Perineal branches
4. Tibial segment of the sciatic nerve
5. Tibial nerve
6. Medial sural cutaneous nerve
7. Sural nerve
8. Tibial nerve in the ankle
9. Medial and lateral plantar nerves
10. Lateral sural cutaneous nerve
11. Common peroneal nerve
12. Common peroneal segment of the sciatic nerve
13. Sciatic nerve
14. Superficial peroneal nerve

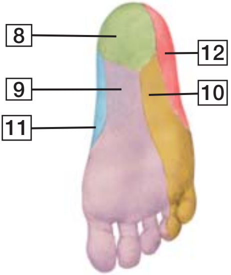
Comment: The superior and inferior gluteal nerves branch proximally, just before the formation of the sciatic nerve. The superior gluteal nerve aids in abduction and inward rotation and some outward rotation of the thigh, as well as in flexion of the upper leg at the hip. The inferior gluteal nerve aids in extension of the thigh at the hip and to outward rotation of the thigh; a lesion results in difficulty climbing stairs and rising from a sitting position. The sciatic nerve (L4–S3) provides motor innervation to the biceps femoris, semitendinosus, and semimembranosus muscles (hamstrings) and regulates flexion of the lower leg; a major lesion results in weakness of leg flexion and weakness of all muscles below the knee. A sciatic nerve lesion also may result in loss of cutaneous sensation on the posterior thigh, posterior and lateral leg, lateral foot, and sole of the foot. A lesion of the posterior femoral cutaneous nerve (S1–S3) results in loss of sensation to the posterior thigh, lateral part of the perineum, and lower portion of the buttock.

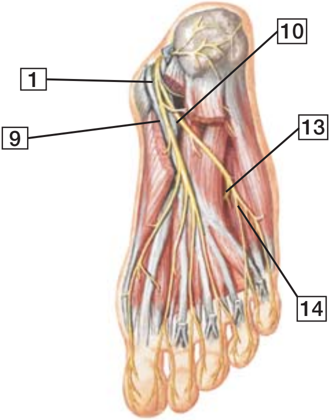
See book 9.34

Tibial Nerve



Cutaneous innervation



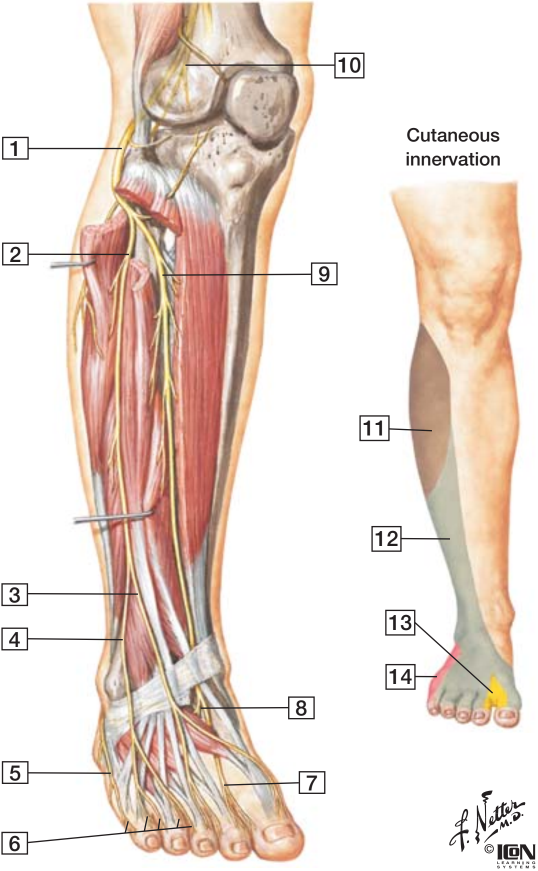


Tibial Nerve

1. Tibial nerve
2. Medial sural cutaneous nerve
3. Sural nerve
4. Lateral calcaneal branch
5. Medial calcaneal branch
6. Lateral sural cutaneous nerve
7. Common peroneal nerve
8. Medial calcaneal branches
9. Medial plantar nerve
10. Lateral plantar nerve
11. Saphenous nerve
12. Sural nerve
13. Deep branch
14. Superficial branch

Comment: The tibial nerve (L4–S2) supplies motor innervation to muscles of plantar flexion and inversion of the foot, as well as toe flexion. A tibial nerve lesion results in weakness of these actions and loss of cutaneous sensation on the lateral calf and in the plantar region of the foot. The tibial nerve is susceptible to an entrapment neuropathy in the tarsal tunnel.

Common Peroneal Nerve



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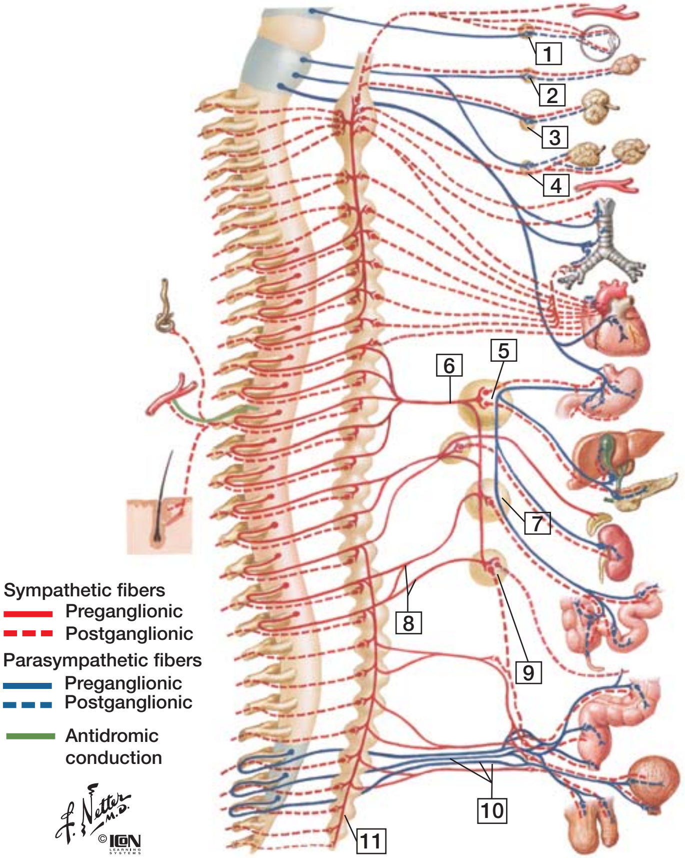
Common Peroneal Nerve

1. Common peroneal nerve
2. Superficial peroneal nerve
3. Medial dorsal cutaneous nerve
4. Intermediate dorsal cutaneous nerve
5. Lateral dorsal cutaneous nerve
6. Proper dorsal digital nerves
7. Medial branch of the deep peroneal nerve
8. Lateral branch of the deep peroneal nerve
9. Deep peroneal nerve
10. Lateral sural cutaneous nerve
11. Lateral sural cutaneous nerve
12. Superficial peroneal nerve
13. Deep peroneal nerve
14. Sural nerve

Comment: The common peroneal nerve (L4–S1) branches into the deep peroneal and superficial peroneal nerves. The common peroneal nerve supplies motor innervation to muscles for dorsiflexion and eversion of the foot and dorsiflexion (extension) of the toes. A lesion of the common peroneal nerve results in loss of these actions, as well as loss of cutaneous sensation on the lateral aspect of the lower leg and the dorsum of the foot.

Schematic of the Autonomic Nervous System



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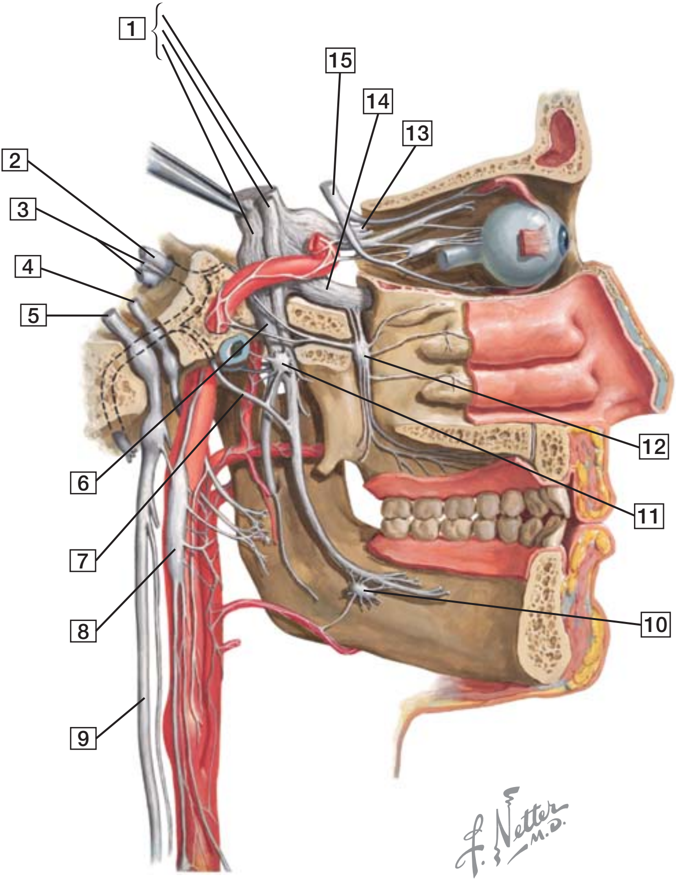
Schematic of the Autonomic Nervous System

1. Ciliary ganglion
2. Pterygopalatine ganglion
3. Otic ganglion
4. Submandibular ganglion
5. Celiac ganglion
6. Greater thoracic splanchnic nerve
7. Superior mesenteric ganglion
8. Lumbar splanchnic nerves
9. Inferior mesenteric ganglion
10. Pelvic splanchnic nerves
11. Sympathetic trunk

Comment: The sympathetic division of the autonomic nervous system (ANS) is derived from neurons in the T1–L2 (thoracolumbar) lateral horn with synapses on chain ganglia (paravertebral) or collateral ganglia (prevertebral). Postganglionic axons distribute to cardiac muscle, smooth muscle, secretory glands, metabolic organs, and immune system tissue. Activation of the sympathetic nervous system can evoke a fight-or-flight response and mobilization for an emergency. The parasympathetic division of the ANS is derived from neurons in the brain stem (CNs III, VII, IX, and X) and from the S2–S4 intermediate gray in the spinal cord, with synapses on intramural (near the target organ) ganglia. Postganglionic axons form neuroeffector junctions with the same target structures as the sympathetics. Sympathetic neuropathies can result in problems such as bradycardia, orthostatic hypotension, inability to sweat, Horner's syndrome, retrograde ejaculation, and gastrointestinal disorders. Parasympathetic neuropathies can result in problems such as tachycardia, a fixed and dilated pupil, urinary dysfunction (bladder weakness), erectile dysfunction, and gastrointestinal disorders.

Autonomic Distribution to the Head and Neck—Medial View

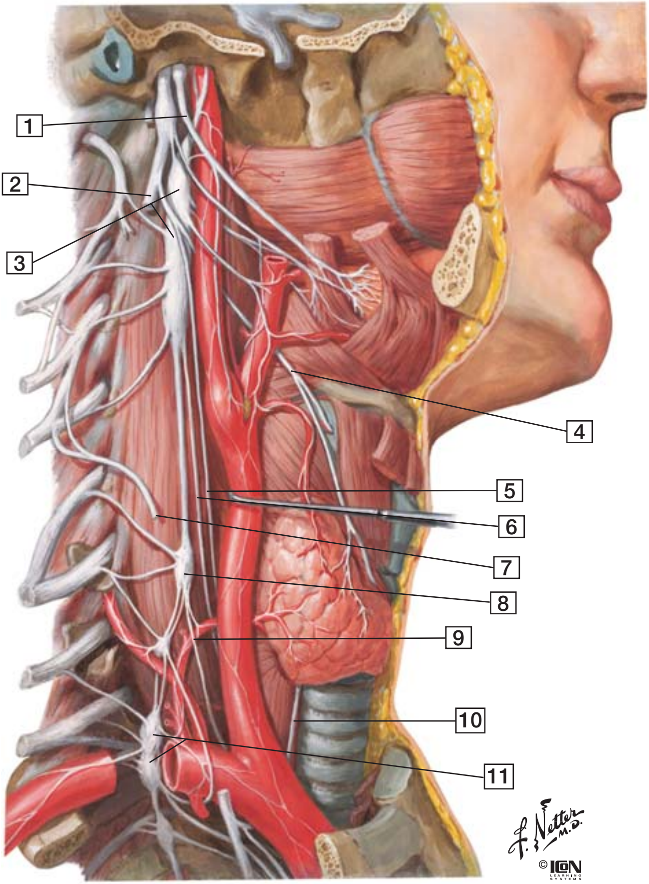


Autonomic Distribution to the Head and Neck—Medial View

1. Trigeminal nerve (CN V)
2. Facial nerve (CN VII)
3. Vestibulocochlear nerve (CN VIII)
4. Glossopharyngeal nerve (CN IX)
5. Vagus nerve (CN X)
6. Mandibular nerve
7. Chorda tympani
8. Superior cervical sympathetic trunk ganglion
9. Vagus nerve (CN X)
10. Submandibular ganglion
11. Otic ganglion
12. Pterygopalatine ganglion
13. Ophthalmic nerve
14. Maxillary nerve
15. Oculomotor nerve (CN III)

Comment: Parasympathetic nerve fibers of CN III, derived from the nucleus of Edinger-Westphal, synapse in the ciliary ganglion, which in turn innervates the pupillary constrictor muscle (efferent limb of the pupillary light reflex) and the ciliary muscle (accommodation to near vision). Parasympathetic nerve fibers of CN VII, derived from the superior salivatory nucleus, synapse in the pterygopalatine ganglion and submandibular ganglion, which in turn innervate the lacrimal glands and nasal mucosa glands, as well as the submandibular and sublingual salivary glands, respectively. Parasympathetic nerve fibers of CN IX, derived from the inferior salivatory nucleus, synapse in the otic ganglion, which in turn innervates the parotid salivary gland.

Autonomic Distribution to the Head and Neck—Lateral View



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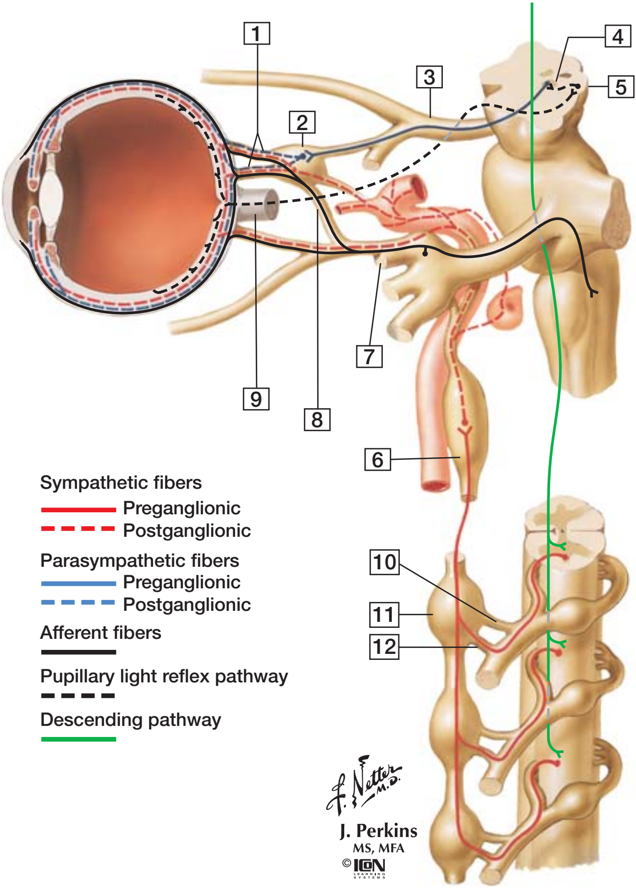
Autonomic Distribution to the Head and Neck—Lateral View

1. Glossopharyngeal nerve (CN IX)
2. Vagus nerve (CN X)
3. Superior cervical sympathetic trunk ganglion
4. Superior laryngeal nerve
5. Superior cervical cardiac branch of the vagus nerve
6. Superior cervical sympathetic cardiac nerve
7. Phrenic nerve
8. Middle cervical sympathetic trunk ganglion
9. Middle cervical sympathetic cardiac nerve
10. Recurrent laryngeal nerve
11. Cervicothoracic (stellate) ganglion

Comment: The superior cervical ganglion (SCG), the most rostral ganglion of the sympathetic chain, supplies structures in the head and neck with noradrenergic postganglionic sympathetic fibers, including the pupillary dilator muscle, blood vessels, sweat glands, the pineal gland, the thymus gland, and the superior tarsal muscle (Muller's muscle). Damage to this ganglion, the preganglionic input to SCG, the cells of origin of the preganglionic nucleus in the T1-T2 intermediolateral cell column, or the descending central fibers in the lateral funiculus of the spinal cord that control the T1-T2 preganglionic outflow to the SCG, results in Horner's syndrome. This syndrome consists of ipsilateral (1) ptosis (drooping eyelid, damage to superior tarsal muscle); (2) miosis (pupillary constriction, from unopposed parasympathetic actions due to damage to pupillary dilation); and (3) anhidrosis (loss of sweating on the face from unresponsive innervation to sweat glands).

Autonomic Distribution to the Eye

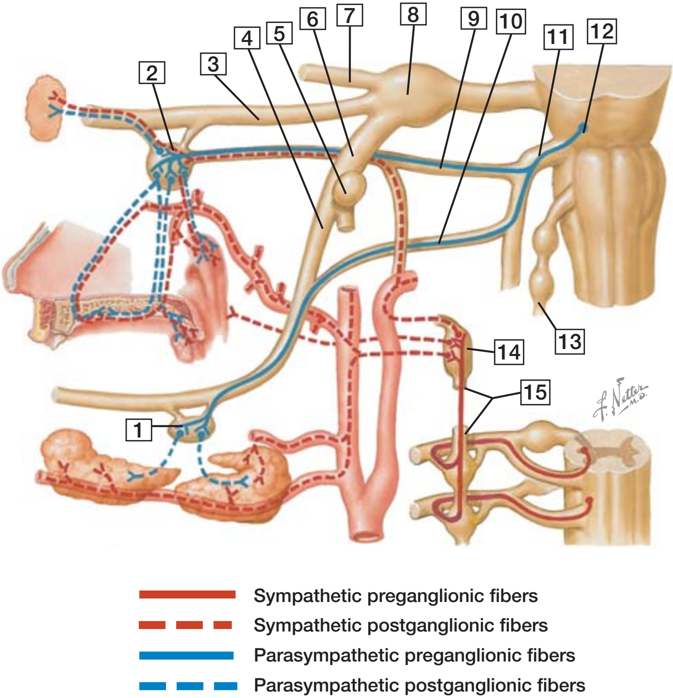


Autonomic Distribution to the Eye

1. Short ciliary nerves
2. Ciliary ganglion
3. Oculomotor nerve (CN III)
4. Nucleus of Edinger-Westphal (CN III)
5. Pretectum
6. Superior cervical sympathetic trunk ganglion
7. Ophthalmic nerve
8. Long ciliary nerve
9. Optic nerve (CN II)
10. Gray ramus communicans
11. First thoracic sympathetic trunk ganglion
12. White ramus communicans

Comment: Smooth muscles controlling the aperture of the pupil are innervated by sympathetic postganglionic noradrenergic fibers (from the superior cervical ganglion to the pupillary dilator muscle) and by parasympathetic postganglionic cholinergic fibers (from the ciliary ganglion, supplied by CN III, to the pupillary constrictor muscle). These sources keep the pupil in balance and responsive to the ambient surroundings. Damage to CN III (e.g., from entrapment against the tentorium cerebelli during transtentorial herniation) results in a fixed, dilated pupil, unresponsive to light. Damage to the sympathetic superior cervical ganglion or its central or preganglionic supply results in a miotic pupil. The ciliary ganglion also supplies the ciliary muscle, which, when contracted, lifts the zonular fibers of the capsule of the lens upward and inward, permitting accommodation for near vision (bunching of the lens for greater refraction of light).

Schematic of Pterygopalatine and Submandibular Ganglia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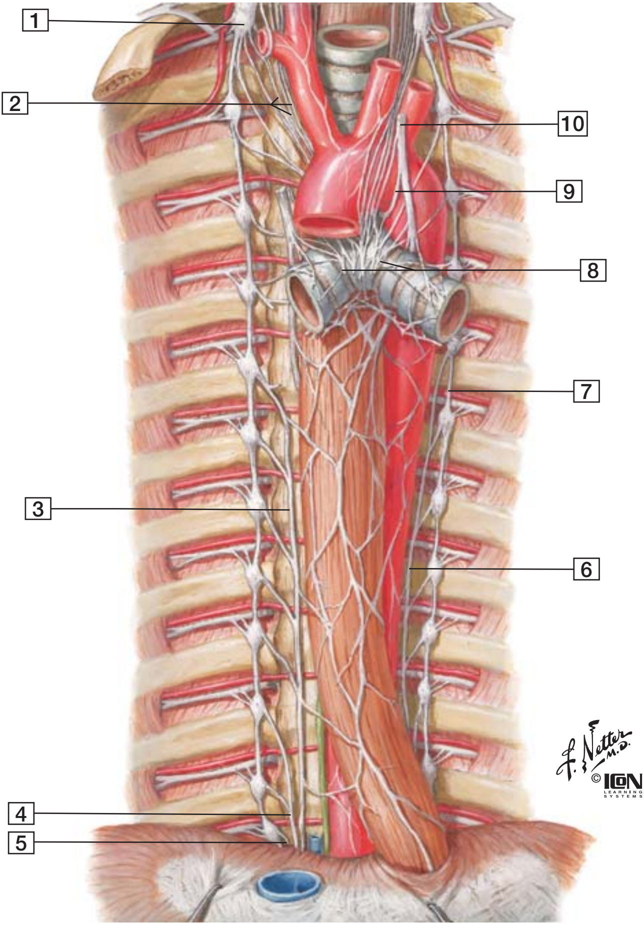
Sympathetic preganglionic fibers
Sympathetic postganglionic fibers
Parasympathetic preganglionic fibers
Parasympathetic postganglionic fibers

Schematic of Pterygopalatine and Submandibular Ganglia

1. Submandibular ganglion
2. Pterygopalatine ganglion
3. Maxillary nerve (CN V2)
4. Lingual nerve
5. Otic ganglion
6. Mandibular nerve (CN V3)
7. Ophthalmic nerve (CN V1)
8. Trigeminal ganglion
9. Greater petrosal nerve
10. Chorda tympani nerve
11. Facial nerve (CN VII)—nervus intermedius
12. Superior salivatory nucleus
13. Glossopharyngeal nerve (CN IX)
14. Superior cervical sympathetic ganglion
15. Sympathetic trunk

Comment: The superior salivatory nucleus (via CN VII) innervates the submandibular ganglion, which supplies the submandibular and sublingual salivary glands. The inferior salivatory nucleus (via CN IX) innervates the otic ganglion, which supplies the parotid salivary gland. These parasympathetic nerve fibers stimulate secretion. The sympathetic postganglionic noradrenergic fibers from the superior cervical ganglion activate contraction of the myoepithelial cells of the salivary ducts. Salivation is important as an initial phase of digestion, preparing food for swallowing. Salivation also aids in speaking. Saliva contains biological mediators and immunoglobulins that provide barrier protection against potentially dangerous organisms that may gain access through the oral cavity.

Thoracic Sympathetic Chain and Splanchnic Nerves

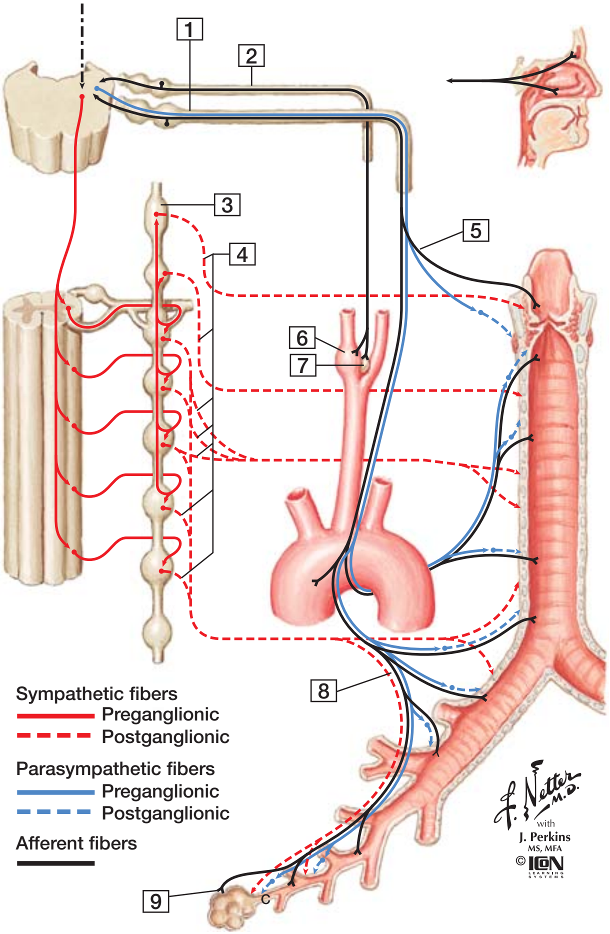


Thoracic Sympathetic Chain and Splanchnic Nerves

1. Cervicothoracic (stellate) ganglion
2. Cervical cardiac nerves (sympathetic and vagal)
3. Right greater thoracic splanchnic nerve
4. Right lesser thoracic splanchnic nerve
5. Right lowest thoracic splanchnic nerve
6. Left greater thoracic splanchnic nerve
7. Left sympathetic trunk
8. Cardiac plexus
9. Left recurrent laryngeal nerve
10. Left vagus nerve

Comment: The sympathetic chain (postganglionic sympathetic neurons, paravertebral ganglia) extends from the neck to the pelvis. Collateral ganglia (prevertebral ganglia) are found associated with the great vessels and distribute short postganglionic fibers to internal target organs. Cholinergic preganglionic neurons from the intermediolateral cell column (T1–L2) send axons through the ventral root and then into the white rami communicantes to either synapse in the sympathetic chain ganglia or pass through the chain and travel via splanchnic nerves (myelinated preganglionic axons) to collateral ganglia. Injury or damage to the ganglia themselves, the preganglionic input to them, or the central control over the preganglionic neurons can result in sympathetic dysfunction to the target organs, resulting in such symptoms as bradycardia, orthostatic hypotension, loss of sweating, and miosis.

Innervation of the Tracheobronchial Tree

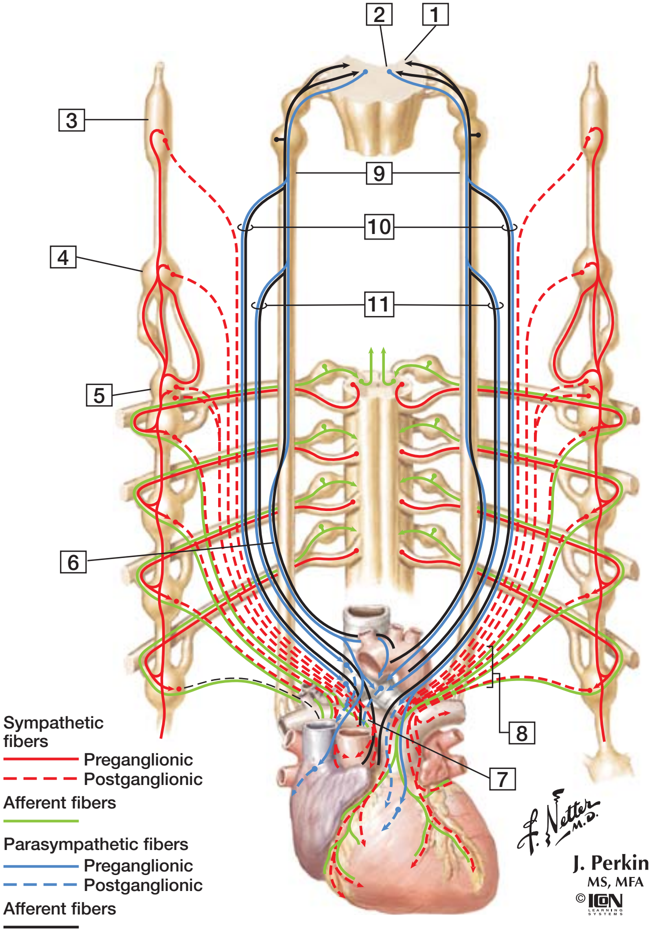


Innervation of the Tracheobronchial Tree

1. Vagus nerve (CN X)
2. Glossopharyngeal nerve (CN IX)
3. Superior cervical sympathetic ganglion
4. Sympathetic nerves
5. Superior laryngeal nerve
6. Carotid sinus
7. Carotid body
8. Pulmonary plexus
9. Stretch receptors (Hering-Breuer reflex)

Comment: Both sympathetic noradrenergic postganglionic fibers and parasympathetic cholinergic postganglionic fibers innervate smooth muscle of the tracheobronchial tree. Sympathetic activation results in bronchodilation and parasympathetic activation results in bronchoconstriction. Some medications for asthma use sympathomimetic agents, whereas others use cholinergic blockade. Neuropeptide-containing nerve fibers, some sensory and some autonomic, also distribute along the epithelium and along the alveoli, where they may influence innate immune reactivity and the production of inflammatory mediators.

Innervation of the Heart



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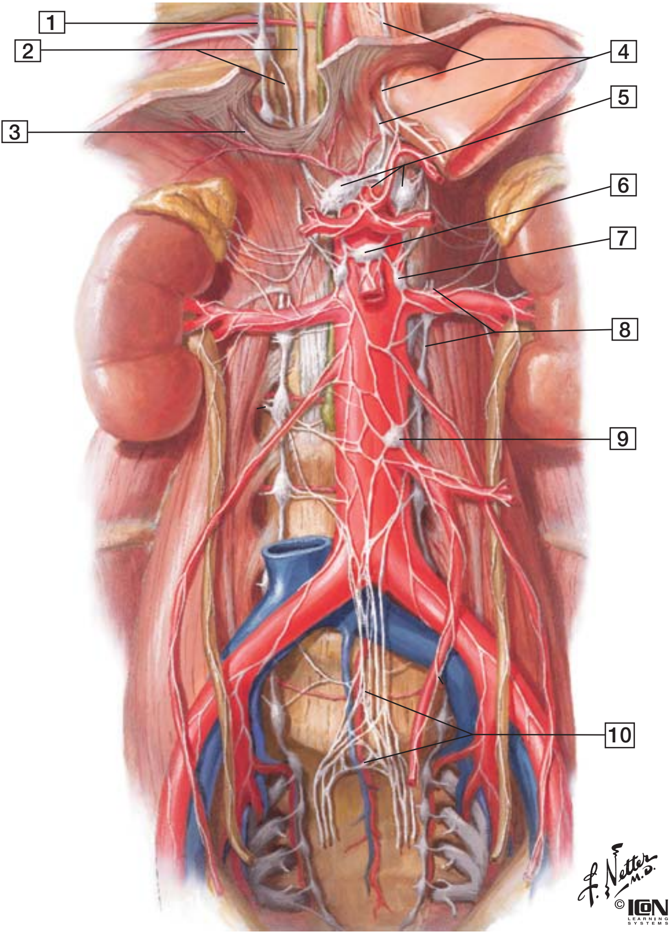
Innervation of the Heart

1. Nucleus of the solitary tract
2. Dorsal vagal nucleus
3. Superior cervical sympathetic trunk ganglion
4. Middle cervical sympathetic trunk ganglion
5. Cervicothoracic (stellate) ganglion
6. Thoracic vagal cardiac branch
7. Cardiac plexus
8. Thoracic sympathetic cardiac nerves
9. Vagus nerves (CN X)
10. Superior cervical vagal cardiac branches
11. Inferior cervical vagal cardiac branches

Comment: Both sympathetic postganglionic noradrenergic and parasympathetic postganglionic cholinergic nerve fibers innervate the heart, including the atria, ventricles, sinoatrial node, and the atrioventricular node and bundle. Sympathetic nerves also distribute along the great vessels and coronary artery. Sympathetic fibers increase the force and rate of cardiac contraction, increase cardiac output, and dilate the coronary arteries. Parasympathetic fibers decrease the force and rate of cardiac contraction and decrease cardiac output. Vagal nerve damage may result in sustained tachycardia. Excessive vagal activity can provoke bradycardia, atrial fibrillation or flutter, ventricular fibrillation, or paroxysmal tachycardia. Sympathetic nerve damage results in severe exercise intolerance, painless myocardial ischemia, possible cardiomyopathy, and on occasion sudden death.

Abdominal Nerves and Ganglia

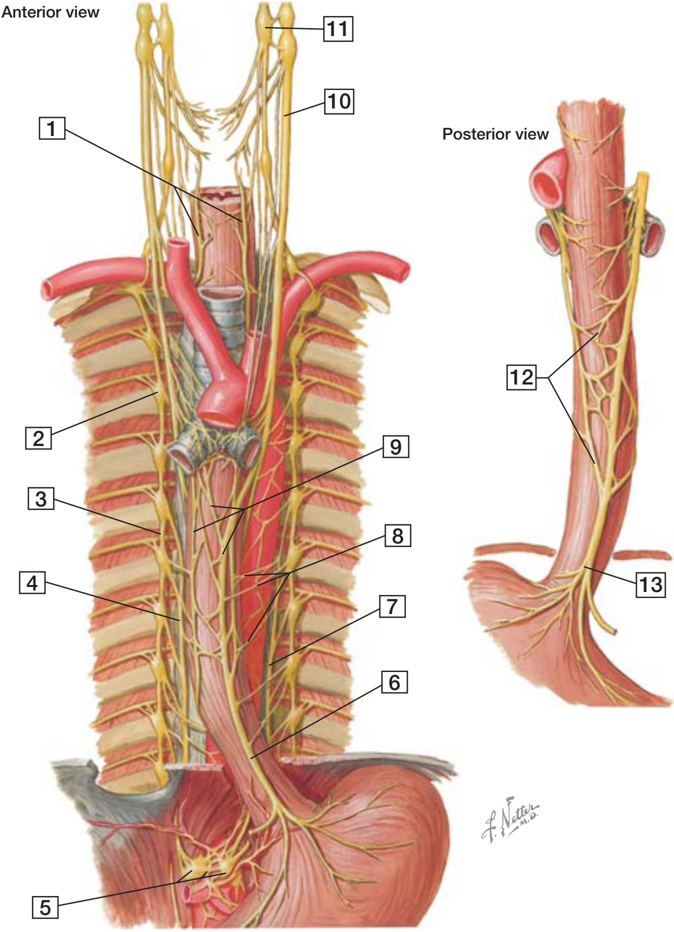


Abdominal Nerves and Ganglia

1. Right sympathetic trunk
2. Right greater and lesser thoracic splanchnic nerves
3. Right phrenic nerves
4. Anterior and posterior vagal trunks
5. Celiac plexus and ganglia
6. Superior mesenteric ganglion
7. Left aorticorenal ganglion
8. Left sympathetic trunk
9. Inferior mesenteric ganglion
10. Superior hypogastric plexus

Comment: The collateral ganglia (celiac, superior and inferior mesenteric, hepatic, aorticorenal, adrenal, superior hypogastric) and the lumbar sympathetic chain supply noradrenergic innervation to the abdomen and pelvis. Parasympathetic vagal fibers and their associated intramural ganglia provide parasympathetic cholinergic innervation to the abdomen and pelvis. Although parasympathetic activation enhances gastrointestinal (GI) contraction and digestion, and sympathetic activation diminishes these functions, the GI tract also contains a large number of intrinsic neurons (enteric nervous system) that regulate peristalsis and absorption. In an autoimmune disorder called *dysautonomic polyneuropathy,* involving both sympathetic and parasympathetic nerves, an individual shows orthostatic hypotension, unresponsive pupillary light responses, paralytic ileus and constipation, bladder dysfunction, diminished sweating, peripheral vasoconstriction, and loss of piloerection.

Nerves of the Esophagus

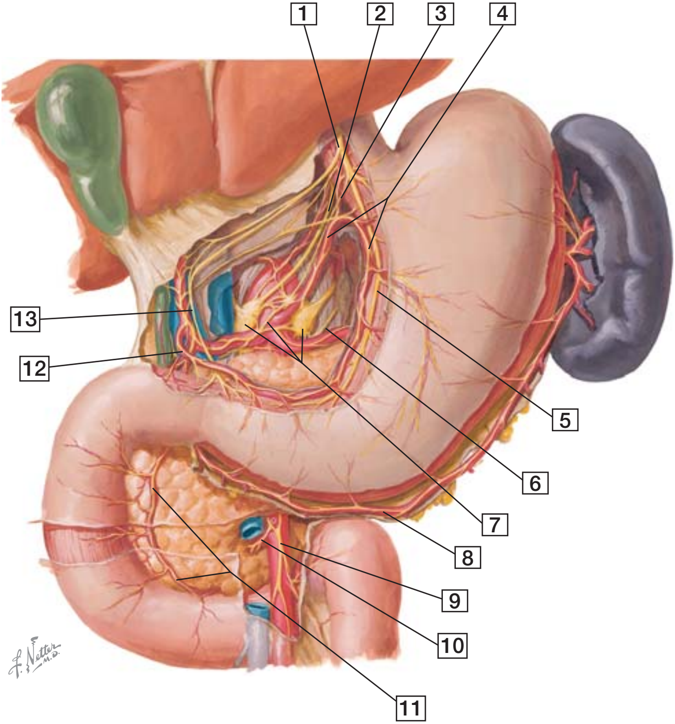


Nerves of the Esophagus

1. Recurrent laryngeal nerves
2. Third thoracic sympathetic ganglion
3. Thoracic sympathetic trunk
4. Right greater splanchnic nerve
5. Celiac plexus and ganglia
6. Anterior vagal trunk
7. Left greater splanchnic nerve
8. Branches to esophageal plexus (from sympathetic trunk, greater splanchnic nerve, thoracic aortic plexus)
9. Esophageal plexus (anterior portion)
10. Vagus nerve (CN X)
11. Superior cervical sympathetic ganglion
12. Esophageal plexus
13. Posterior vagal trunk

Comment: The sensory stimuli that initiate swallowing derive mainly from CN IX (nucleus solitarius). Movement of food through the cricopharyngeal sphincter into the proximal esophagus is controlled by vagal fibers from the dorsal (motor) nucleus of CN X. Movement of food through the esophagus is regulated by vagal fibers from the dorsal (motor) nucleus of CN X via synapses on ganglia of the myenteric plexus of the esophagus. This plexus controls peristalsis through alternating relaxation and contraction of esophageal muscles. Food moves into the stomach through the lower esophageal sphincter, which relaxes when nitric oxide and vasoactive intestinal peptide are released from some neurons in the myenteric plexus.

Nerves of the Stomach and Duodenum

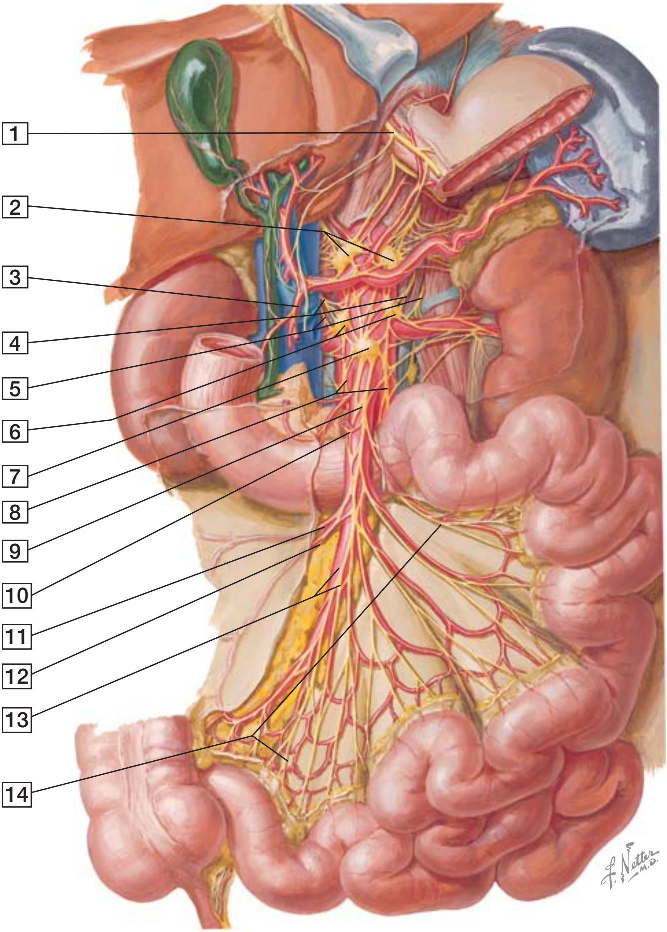


Nerves of the Stomach and Duodenum

1. Anterior vagal trunk
2. Celiac branch of the posterior vagal trunk
3. Celiac branch of the anterior vagal trunk
4. Left gastric artery and plexus
5. Anterior gastric branch of the anterior vagal trunk
6. Left lesser splanchnic nerve
7. Celiac ganglia and plexus
8. Plexus on gastro-omental (gastroepiploic) arteries
9. Superior mesenteric artery and plexus
10. Plexus on inferior pancreaticoduodenal artery
11. Plexus on anterior superior and anterior inferior pancreaticoduodenal arteries
12. Right gastric artery and plexus
13. Vagal branch from hepatic plexus to pyloric part of stomach

Comment: Both sympathetic and parasympathetic postganglionic nerve fibers distribute to the stomach and proximal duodenum through specific splanchnic nerves and branches of the vagus nerve. Sympathetic fibers decrease peristalsis and secretomotor activities. Parasympathetic fibers increase peristalsis and secretomotor activities such as gastrin and hydrochloric acid secretion and relax associated sphincters, promoting movement of food.

Nerves of the Small Intestine

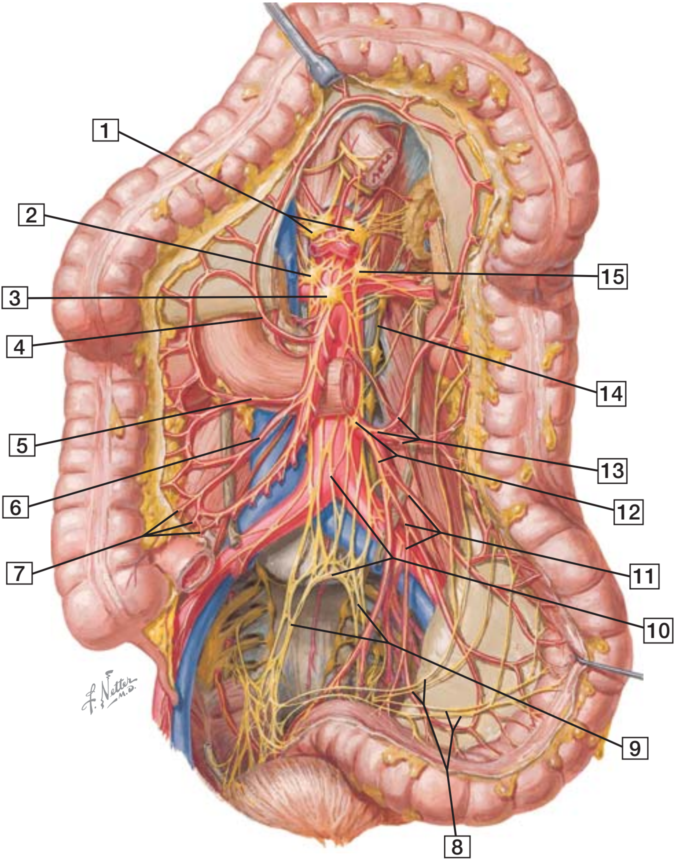


Nerves of the Small Intestine

1. Anterior vagal trunk
2. Celiac ganglia and plexus
3. Gastroduodenal artery and plexus
4. Lesser splanchnic nerves
5. Least splanchnic nerves
6. Aorticorenal ganglia
7. Superior mesenteric ganglion
8. Intermesenteric (aortic) plexus
9. Superior mesenteric artery and plexus
10. Middle colic artery and plexus
11. Right colic artery and plexus
12. Ileocolic artery and plexus
13. Superior mesenteric artery and plexus
14. Mesenteric branches

Comment: Autonomic innervation of the small and large intestines is supplied by extrinsic sympathetic and parasympathetic postganglionic nerve fibers. Sympathetic innervation is supplied by T5–L2 preganglionic neurons through collateral ganglia (superior and inferior mesenteric, celiac). Parasympathetic innervation is supplied from vagus nerve (CN X) and from S2–S4 preganglionic neurons (via pelvic splanchnic nerves) through intramural ganglia. Sympathetic nerve fibers generally decrease peristalsis and inhibit secretomotor functions. Parasympathetic fibers increase peristalsis, relax involuntary sphincters, and promote secretomotor activities.

Nerves of the Large Intestine

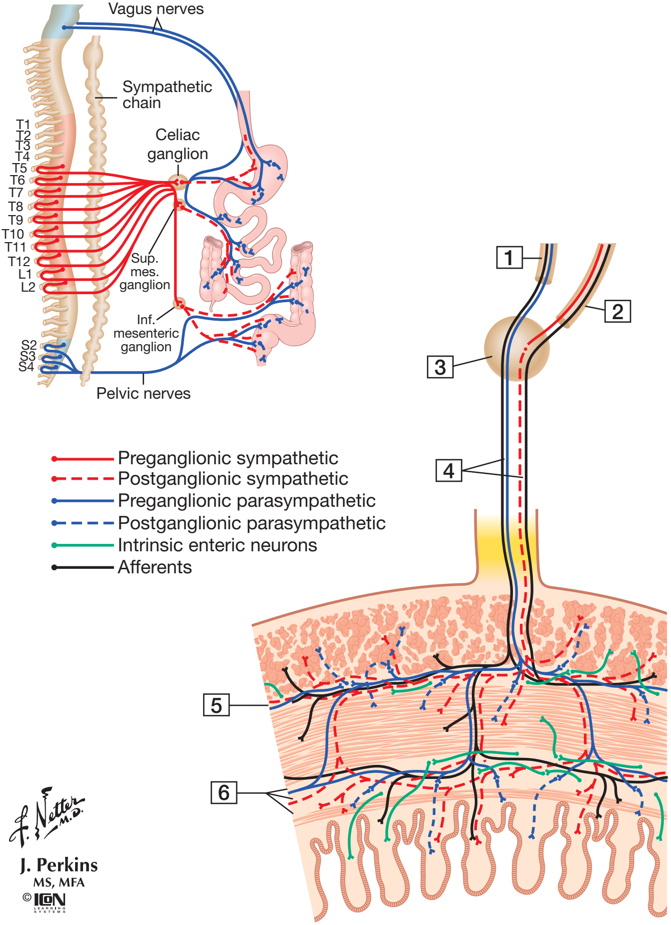


Nerves of the Large Intestine

1. Celiac ganglion and plexus
2. Right aorticorenal ganglion
3. Superior mesenteric ganglion
4. Middle colic artery and plexus
5. Right colic artery and plexus
6. Ileocolic artery and plexus
7. Cecal and appendicular arteries and plexus
8. Nerves from inferior hypogastric plexuses to sigmoid colon, descending colon, and left colic (splenic) flexure
9. Right and left hypogastric nerves
10. Superior hypogastric plexus
11. Sigmoid arteries and plexuses
12. Inferior mesenteric ganglion, artery, and plexus
13. Left colic artery and plexus
14. Left lumbar sympathetic trunk
15. Left aorticorenal ganglion

Comment: The extensive autonomic innervation of the small and large intestines derives from collateral ganglia of the sympathetic nervous system and intramural ganglia of the parasympathetic nervous system. These nerve fibers affect smooth muscle fibers and secretomotor functions of the gastrointestinal (GI) tract directly. The autonomic input to the intestines also regulates large networks of intrinsic enteric neurons, which can act autonomously, even in the absence of direct autonomic input. Thus, the enteric nervous system has two levels of control, intrinsic and extrinsic (from autonomic postganglionic neurons). Autonomic neuropathies, such as those seen in patients with diabetes, can affect the GI tract. Diabetic autonomic neuropathy most often results in constipation, requiring pharmaceutical treatment and use of high-fiber agents. However, persistent diarrhea also may occur, requiring treatment for slowing of secretomotor function.

Enteric Nervous System



— Preganglionic sympathetic
- - - Postganglionic sympathetic
— Preganglionic parasympathetic
- - - Postganglionic parasympathetic
— Intrinsic enteric neurons
— Afferents

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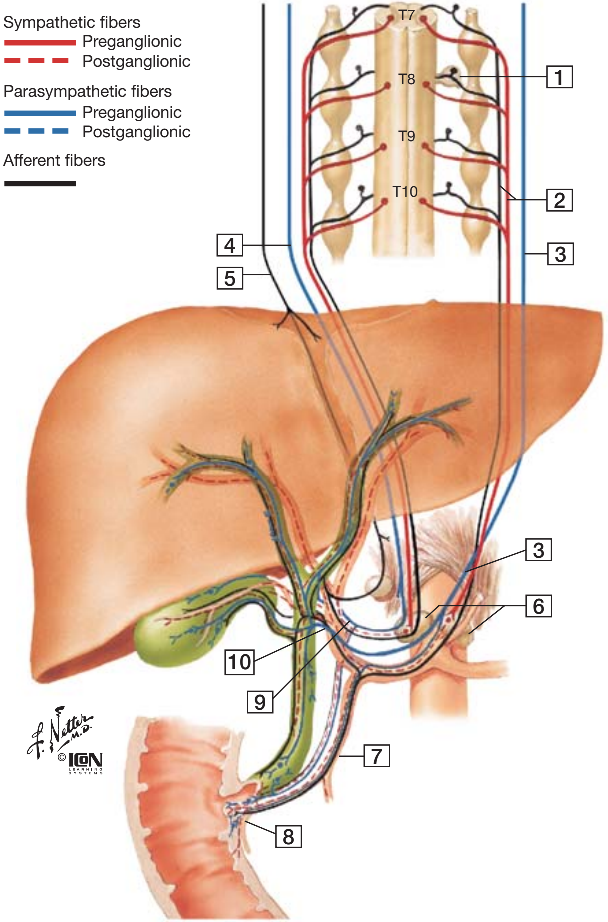
Enteric Nervous System

1. Vagus nerve
2. Splanchnic nerve
3. Celiac or superior mesenteric ganglion (collateral ganglion)
4. Mesenteric nerves
5. Myenteric plexus (Auerbach's plexus)
6. Submucous (submucosal) plexus (Meissner's plexus)

Comment: The enteric nervous system consists of approximately 100 million neurons arranged principally in the submucosal (Auerbach's) and myenteric (Auerbach's) plexuses. Enteric neurons and their plexuses regulate peristaltic responses (which may occur without extrinsic control), pacemaker activity, and automated secretory activity. Extrinsic (autonomic) input contacts only a small number of these enteric neurons but may help to coordinate their activity. If the intrinsic enteric neurons (neural crest origin) fail to develop or migrate properly, a disorder called Hirschsprung's disease (chronic megacolon) occurs. The proper circuitry for peristalsis, pacemaker activity, and secretomotor functions is not present, and the autonomic extrinsic nerves cannot achieve effective coordination of neural circuitry that is mainly absent. The lack of peristalsis and smooth muscle tone results in megacolon, with hypertrophy and distention.

Autonomic Innervation of the Liver and Biliary Tract

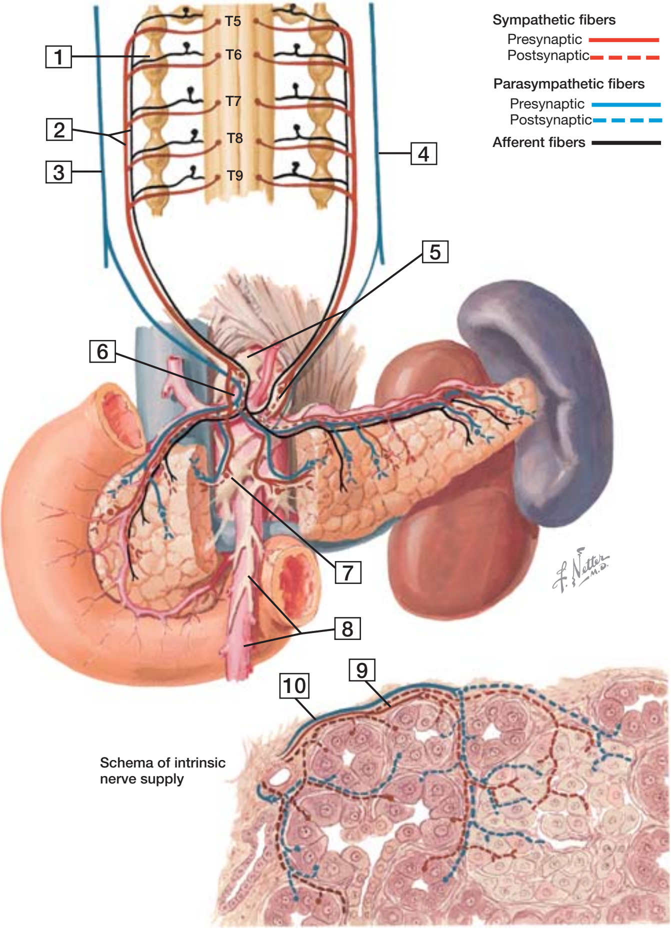


Autonomic Innervation of the Liver and Biliary Tract

1. Dorsal root ganglion
2. Left greater thoracic splanchnic nerve
3. Anterior vagal trunk
4. Posterior vagal trunk
5. Right phrenic nerve
6. Celiac ganglion
7. Gastroduodenal artery and plexus
8. Sphincter ampullae
9. Posterior hepatic plexus
10. Anterior hepatic plexus

Comment: Sympathetic nerve fibers to the liver arise from T7-T10 segments of the spinal cord and distribute mainly via the celiac ganglion and its associated plexuses. Parasympathetic nerve fibers to the liver arise from the abdominal vagus nerve and distribute through intramural ganglia. Postganglionic noradrenergic sympathetic nerve terminals end immediately adjacent to hepatocytes; released norepinephrine stimulates glycogenolysis for activational (fight-or-flight) responses and can stimulate gluconeogenesis for subsequent production of a glucose source. Collectively, autonomic input to the liver assists in vascular, secretory, and phagocytic processes in the liver. Autonomic fibers also innervate the ampullae and sphincter of the choledochal duct; sympathetics cause contraction of the sphincter and dilation of the gallbladder, whereas parasympathetics cause opening of the sphincters and contraction of the gallbladder. Autonomic neuropathy affecting the gallbladder may result in atonic smooth muscle responses, with the development of gallstones (especially in individuals with hypercholesterolemia) and diarrhea.

Autonomic Innervation of the Pancreas

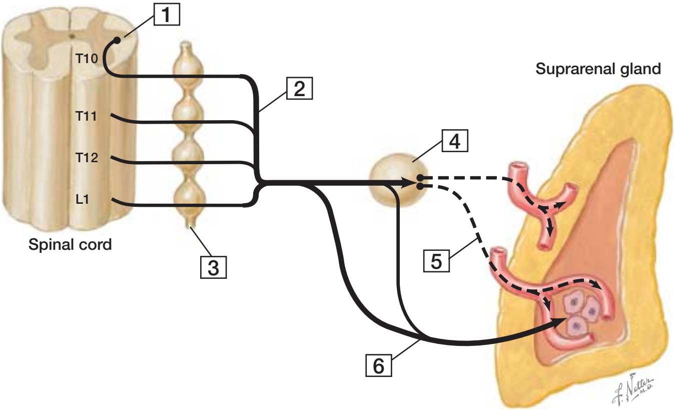


Schema of intrinsic nerve supply

Autonomic Innervation of the Pancreas

1. Right sympathetic trunk
2. Right greater thoracic splanchnic nerve
3. Posterior vagal trunk
4. Anterior vagal trunk
5. Celiac ganglia
6. Celiac trunk
7. Superior mesenteric ganglion
8. Superior mesenteric artery and plexus
9. Sympathetic input to the pancreas
10. Parasympathetic input to the pancreas

Comment: Pancreatic secretion is regulated by both endocrine and neural processes. Sympathetic input derives from T5–T9 segments of the spinal cord and is distributed mainly via the celiac ganglion. Parasympathetic input derives from the subdiaphragmatic vagus nerve and is distributed via intramural ganglia. The endocrine component of the pancreas secretes several vital endocrine products, including glucagon, insulin, somatostatin, and pancreatic polypeptide. Acetylcholine from the parasympathetic nerves stimulates insulin secretion by pancreatic islet cells, while norepinephrine from sympathetic nerves (and hormonal epinephrine) inhibits insulin secretion. Acetylcholine also can stimulate a variety of hormones from the pancreas.


1
T10
T11
T12
L1
Spinal cord
2
3
4
5
6
Suprarenal gland
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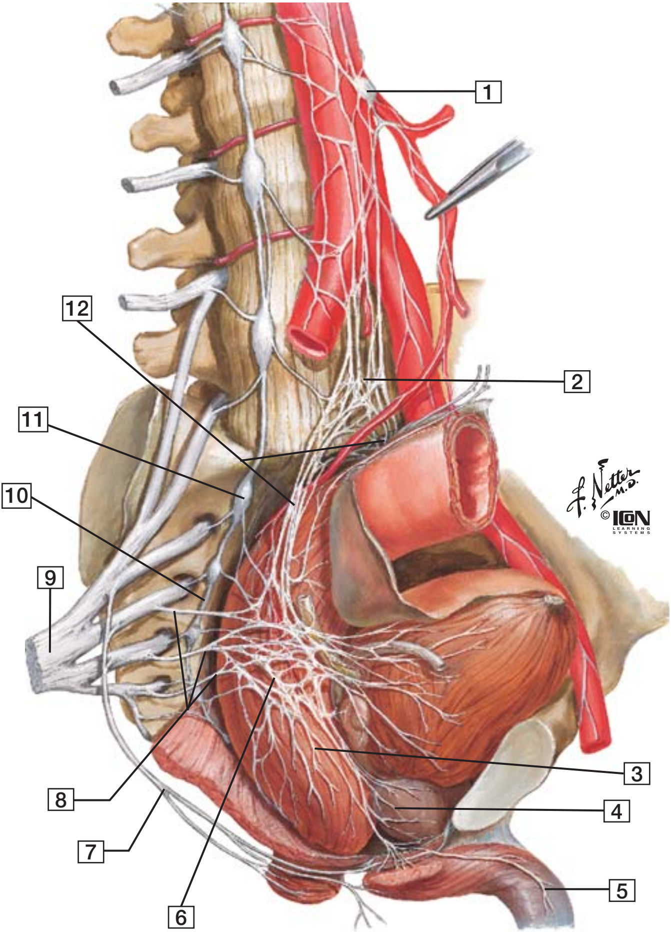
Innervation of the Adrenal Gland

1. Intermediolateral cell column (lateral horn)
2. Abdominopelvic splanchnic nerves
3. Sympathetic trunk
4. Celiac, aorticorenal, and renal ganglia (collateral ganglia)
5. Postganglionic sympathetic nerve fibers to blood vessels
6. Preganglionic sympathetic fibers innervating adrenal medullary chromaffin cells

Comment: Sympathetic input to the adrenal gland derives from T10–L1 segments of the spinal cord and is distributed via splanchnic nerves directly into the adrenal medulla, where preganglionic nerve fibers form neuroeffector junctions with chromaffin cells. These adrenal medullary chromaffin cells are neural in origin, derived from neural crest. Sympathetic preganglionic stimulation results in secretion of epinephrine (80%) and norepinephrine (20%) from these chromaffin cells into the systemic circulation. These neurotransmitters act on alpha- and beta-adrenergic receptors on target cells, aiding in the fight-or-flight activational response, and also may be taken up by high-affinity uptake carriers into the postganglionic noradrenergic nerve terminals and subsequently preferentially released and used to augment the sympathetic activation. Endocrine input to the chromaffin cells from the glucocorticoid-producing cells in the adrenal cortex, via an adrenal portal vascular system, induces in chromaffin cells the expression of phenylethanolamine *N*-methyl transferase (PNMT), the enzyme that converts norepinephrine to epinephrine, thus accounting for the predominant output of epinephrine from the chromaffin cells. Postganglionic nerve fibers from collateral ganglia also provide sympathetic innervation to blood vessels supplying the adrenal gland.

Autonomic Pelvic Nerves and Ganglia

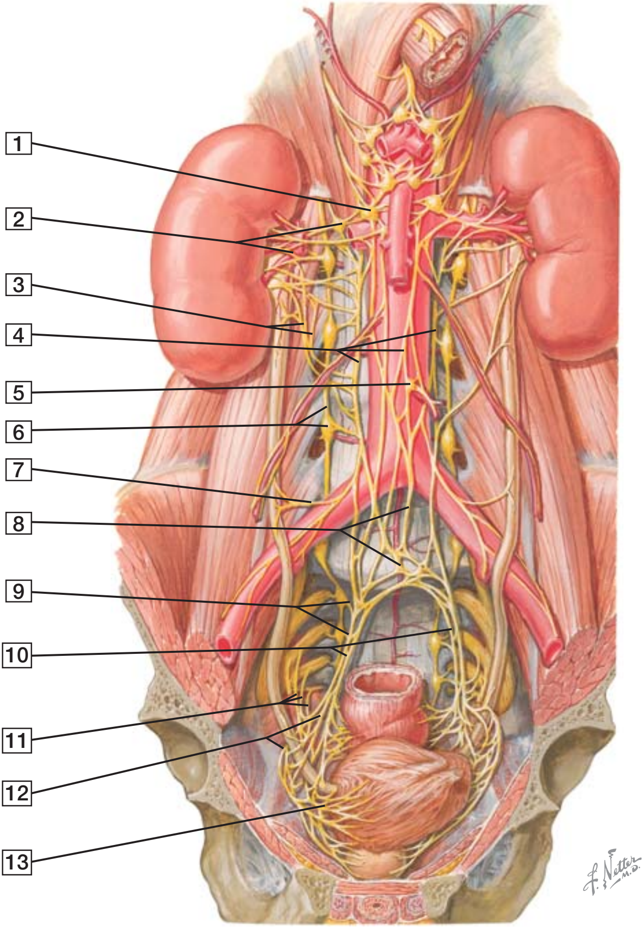


Autonomic Pelvic Nerves and Ganglia

1. Inferior mesenteric ganglion
2. Superior hypogastric plexus (presacral nerve)
3. Inferior rectal plexus
4. Prostatic plexus
5. Dorsal nerve of the penis
6. Right inferior hypogastric (pelvic) plexus
7. Pudendal nerve
8. Pelvic splanchnic nerves
9. Sacral plexus
10. Sacral part of the sympathetic trunk
11. First sacral sympathetic trunk ganglion
12. Right and left hypogastric nerves

Comment: The pelvic nerves and ganglia contain both sympathetic and parasympathetic components. The sympathetic supply derives from the sympathetic chain ganglia and the superior hypogastric plexus; the parasympathetic supply derives from the S2–S4 segments of the spinal cord via pelvic splanchnic nerves and the inferior hypogastric plexuses and ends in intramural ganglia that supply the pelvic viscera. Damage to pelvic parasympathetic nerves may produce a flaccid bladder with overflow incontinence and erectile dysfunction. Sexual function depends on coordinated activity of both the parasympathetics and sympathetics. Parasympathetic nerves are necessary for proper erectile function and sympathetics are necessary for aspects of ejaculation (sympathetic lesion results in retrograde ejaculation). In addition, administration of beta blockers may also result in erectile dysfunction.

Nerves of the Kidneys, Ureters, and Urinary Bladder



Nerves of the Kidneys, Ureters, and Urinary Bladder

1. Aorticorenal ganglion
2. Renal plexus and ganglion
3. Renal and upper ureteric branches from intermesenteric plexus
4. Intermesenteric (aortic) plexus
5. Inferior mesenteric ganglion
6. Sympathetic trunk and ganglion
7. Middle ureteric branch
8. Superior hypogastric plexus
9. Sacral splanchnic nerves
10. Hypogastric nerves
11. Pelvic splanchnic nerves
12. Inferior hypogastric (pelvic) plexus to lower ureter
13. Vesical plexus

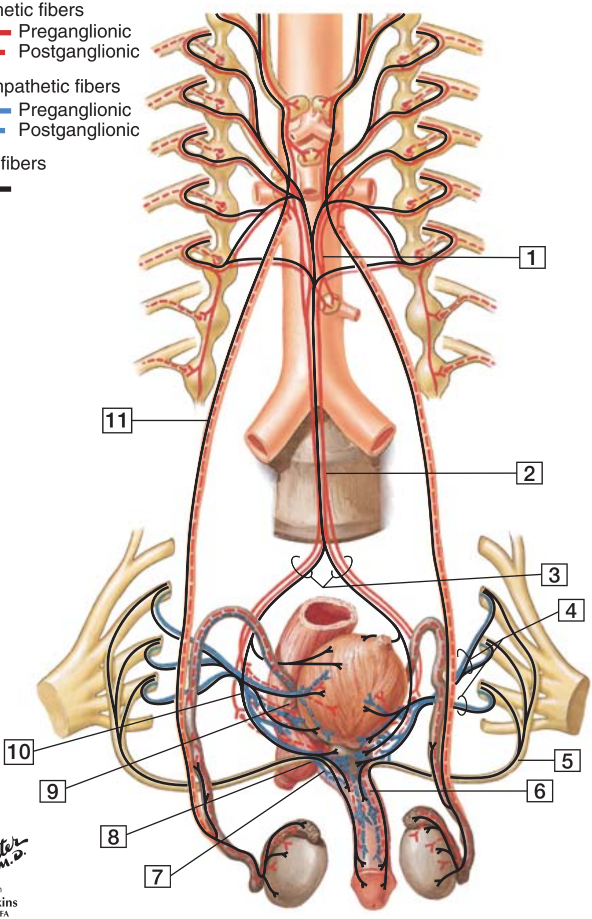
Comment: Sympathetic innervation to the kidney derives from T10–L1 segments of the spinal cord via lower thoracic and upper lumbar splanchnic nerves to celiac and aorticorenal ganglia. These nerves distribute to the upper ureter and to renal, pelvic, calyceal, and segmental branches of the renal vessels. Parasympathetic innervation derives from the vagus nerve and pelvic splanchnic nerves to the renal ganglia. Sympathetic activation stimulates the renin-angiotensin-aldosterone system (elevated blood pressure, decreased glomerular filtration rate, reduced Na^+Cl^- secretion) and stimulates contraction of the ureters. Parasympathetic activation relaxes smooth muscles within the pelvis, calyces, and ureters. Sympathetic innervation of the bladder, derived from L1–L2 segments via the hypogastric plexus, relaxes the detrusor muscle and causes the trigone and internal sphincter of the bladder to contract. Parasympathetic innervation of the bladder, derived from S2–S4 segments via intramural ganglia, results in contraction of the detrusor muscle and relaxation of the trigone and internal sphincter, stimulating bladder emptying. Sensory fibers also are present in the bladder, and they signal stretching, initiating the sensation of the need to empty the bladder.

Innervation of the Male Reproductive Organs

Sympathetic fibers
▬▬ Preganglionic
- - - Postganglionic

Parasympathetic fibers
▬▬ Preganglionic
- - - Postganglionic

Afferent fibers
▬▬



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Innervation of the Male Reproductive Organs

1. Intermesenteric plexus
2. Superior hypogastric plexus (presacral nerve)
3. Hypogastric nerves
4. Left pelvic splanchnic nerves
5. Pudendal nerve
6. Dorsal nerve of the penis
7. Penile cavernous nerves
8. Prostatic plexus
9. Vesical plexus
10. Inferior hypogastric (pelvic) plexus
11. Right testicular artery and plexus

Comment: Sympathetic innervation to male reproductive organs derives from T10–L2 segments of the spinal cord via thoracic and upper lumbar splanchnic nerves to the superior hypogastric plexus. Parasympathetic innervation derives from S2–S4 segments of the spinal cord via pelvic splanchnic nerves to the inferior hypogastric plexus. Sympathetic nerves cause contraction of the vas deferens and the prostatic capsule and contract the sphincter to the bladder, preventing retrograde ejaculation. In the corpora cavernosa of the penis, the sympathetic nerves also contribute to vascular responses related to erection. Parasympathetic nerves regulate the vascular dilation that initiates and maintains penile erection.

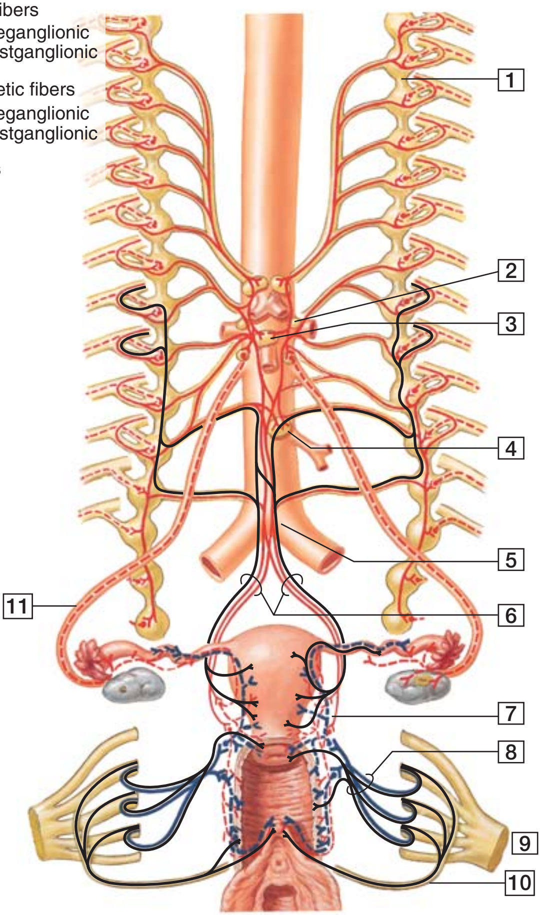
See book 9.70

Innervation of the Female Reproductive Organs

Sympathetic fibers
🟥━━ Preganglionic
🟥╍╍ Postganglionic

Parasympathetic fibers
🟦━━ Preganglionic
🟦╍╍ Postganglionic

Afferent fibers
━━



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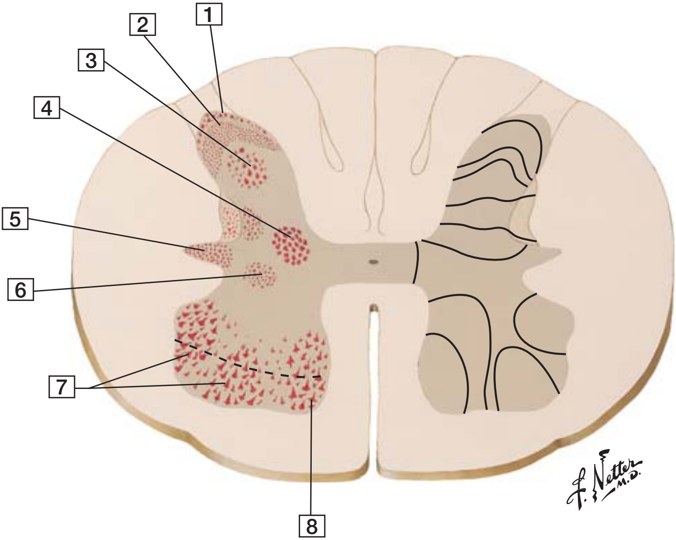
Innervation of the Female Reproductive Organs

1. Left sympathetic trunk
2. Aorticorenal ganglion
3. Superior mesenteric ganglion
4. Inferior mesenteric ganglion
5. Superior hypogastric plexus (presacral nerve)
6. Hypogastric nerves
7. Uterovaginal plexus
8. Pelvic splanchnic nerves
9. Sacral plexus
10. Pudendal nerve
11. Ovarian artery and plexus

Comment: Sympathetic innervation to female reproductive organs derives from T10–L2 segments of the spinal cord via thoracic and upper lumbar splanchnic nerves to the superior hypogastric plexus. Parasympathetic innervation derives from the S2–S4 segments of the spinal cord via pelvic splanchnic nerves to the inferior hypogastric plexus. Sympathetic nerve activation stimulates contraction of the uterus, but hormonal influences and neurotransmitter receptor expression are important components to permitting such actions. Sympathetic nerves also supply the vaginal arteries, the vestibular glands, and clitoral erectile tissue. Parasympathetic nerves supply the muscular and mucous coats of the vagina and urethra, stimulate erectile tissue of the clitoris, and supply the vestibular glands. Autonomic neuropathy involving innervation to the female reproductive organs may cause dry, atrophic vaginal walls with little lubrication, resulting in dyspareunia (painful intercourse).

Cytoarchitecture of the Spinal Cord Gray Matter



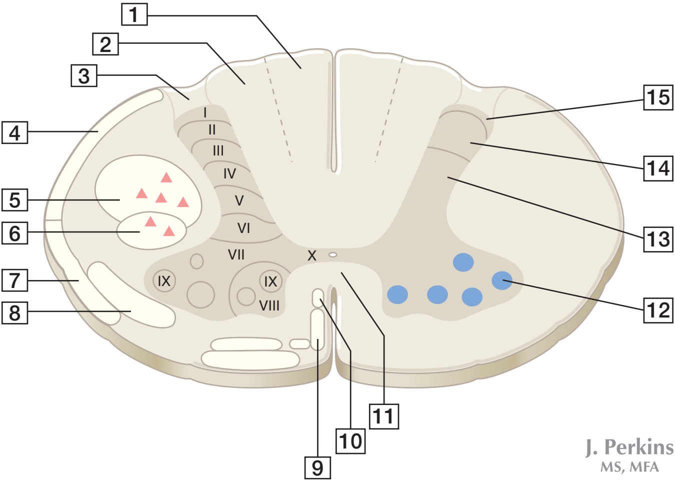
Cytoarchitecture of the Spinal Cord Gray Matter

1. Nucleus posterior marginalis (marginal zone)
2. Substantia gelatinosa (lamina II)
3. Nucleus proprius of the dorsal (posterior) horn
4. Nucleus dorsalis (Clarke's column)
5. Intermediolateral cell column
6. Intermediomedial cell column
7. Motor neurons of limbs
8. Motor neurons of trunk and neck

Comment: Spinal cord neurons are found in the centrally located gray matter, subdivided into a dorsal (posterior) horn (DH), intermediate gray (IG), and a ventral (anterior) horn (VH). The DH processes extensive sensory information (arriving via the dorsal roots), including pain, temperature sensation, and some mechanoreception. Lamina I and V process, for further conveyance into the spinothalamic tract. A cascading system of DH and IG neurons process sensation associated with agonizing, long-lasting pain via the spino-reticular system; both the spinothalamic and spino-reticular systems travel in the contralateral anterolateral funiculus. Additional secondary sensory neurons in the DH and IG (e.g., Clarke's nucleus) receive information from the joints, muscles, and deep tissues and convey this information to the cerebellum. The IG contains preganglionic neurons of the sympathetic (T1–L2 intermediolateral cell column in the lateral horn) and parasympathetic (S2–S4 intermediomedial cell column in the intermediate gray) systems. These neurons send efferent myelinated axons through the ventral root to synapse in sympathetic chain ganglia or collateral ganglia or parasympathetic intramural ganglia. Lower motor neurons are found in localized clusters in the VH (limbs lateral, trunk and body medial) and send their axons into the ventral roots and then into peripheral nerves, from which they distribute to skeletal muscles.

C7 Spinal Cord Cross Section

▲ Descending monoamine axons
(noradrenergic, serotonergic)
Descending fibers from hypothalamus
and brain stem to spinal cord



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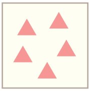
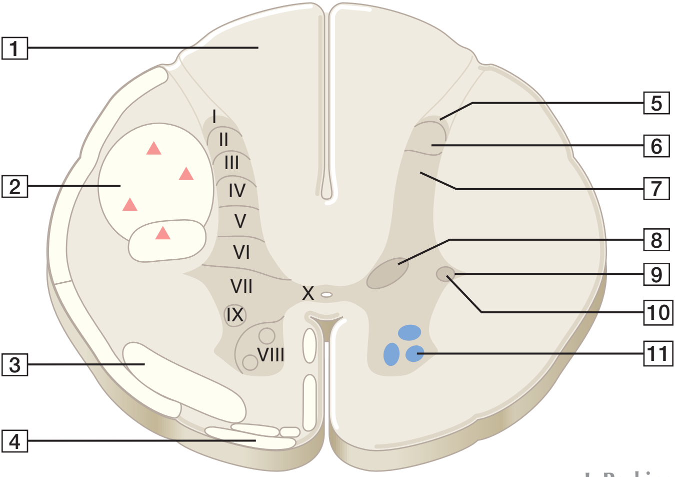
C7 Spinal Cord Cross Section

1. Fasciculus gracilis
2. Fasciculus cuneatus
3. Dorsolateral fasciculus (Lissauer's zone)
4. Dorsal spinocerebellar tract
5. Lateral corticospinal tract
6. Rubrospinal tract
7. Ventral spinocerebellar tract
8. Anterolateral system (spinothalamic and spinoreticular tracts)
9. Anterior corticospinal tract
10. Medial longitudinal fasciculus
11. Anterior white commissure
12. Lower motor neurons in anterior horn
13. Nucleus proprius
14. Substantia gelatinosa
15. Marginal zone

Comment: White matter is found in outer regions (funiculi) of the spinal cord, the dorsal, lateral, and ventral (anterior) funiculi. White matter increases in absolute amount from caudal to rostral. The dorsal funiculus (columns) contains only fasciculus gracilis below T6; fasciculus cuneatus joins the dorsal columns above T6. The anterolateral system increases from caudal to rostral. The descending upper motor neuron (UMN) pathways in the lateral funiculus diminish from rostral to caudal, reflecting synapses in successive segments of the ventral horn. The dorsal funiculus contains mainly epicritic primary sensory information (fine discriminative touch, vibratory sensation, joint position sense). The lateral funiculus contains descending UMN pathways (corticospinal, rubrospinal tracts); spinocerebellar pathways (dorsal and ventral); and descending central autonomic axons from the hypothalamus and brain stem. Damage to the lateral funiculus on one side results in ipsilateral paresis below the level of the lesion (initially flaccid, resolving to spasticity with hypertonus and hyperreflexia), and Horner's syndrome (ptosis, miosis, anhidrosis of the face) with damage above C8.



Descending monoamine axons
(noradrenergic, serotonergic)
Descending fibers from hypothalamus
and brain stem to spinal cord



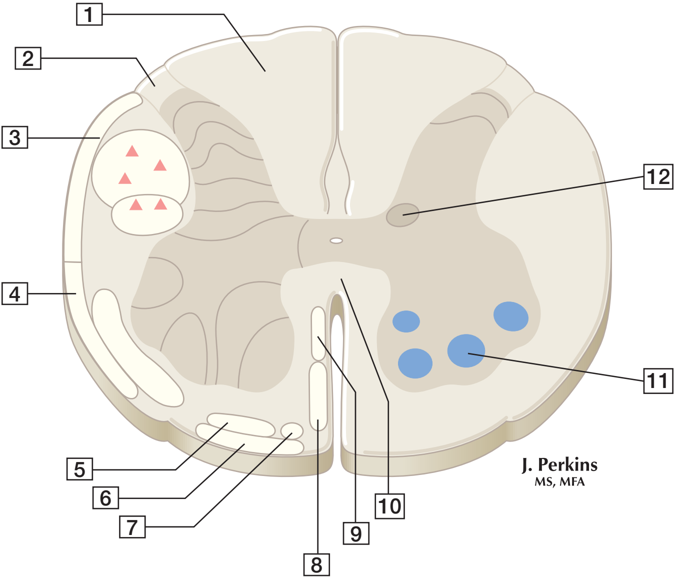
T8 Spinal Cord Cross Section

1. Fasciculus gracilis
2. Lateral corticospinal tract
3. Anterolateral system
4. Lateral vestibulospinal tract
5. Marginal zone
6. Substantia gelatinosa
7. Nucleus proprius
8. Nucleus dorsalis of Clarke
9. Lateral horn
10. Intermediolateral cell column
11. Lower motor neurons in anterior horn

Comment: An injury that damages one side of the spinal cord is known as the Brown-Séquard syndrome. It consists of (1) flaccid paralysis of ipsilateral muscles innervated by lower motor neurons (LMNs) at the level of damage; (2) spastic paralysis (after 1 week or so) of ipsilateral muscles innervated by LMNs below the level of damage, deprived of their upper motor neuron (UMN) control due to lateral funiculus damage; (3) loss of epicritic sensation ipsilaterally below the level of the lesion due to disruption of the dorsal columns; (4) loss of protopathic sensation contralaterally below the level of the lesion due to disruption of the crossed anterolateral system; and (5) Horner's syndrome (ptosis, miosis, anhidrosis of the face) ipsilaterally if the injury is at the C7 level or higher. A developmental defect may occur that forms a syrinx (or cavitation) in the region of the central canal of the spinal cord, a zone that was originally the central core of the developing ventricular system; this condition is called syringomyelia. This syrinx may expand to damage crossing axons of the anterior white commissure, resulting in bilateral loss (sometimes patchy) of pain and temperature sensation due to disruption of the crossing spinothalamic and spinoreticular axons, with fully preserved epicritic sensation.

L3 Spinal Cord Cross Section

▲ Descending monoamine axons (noradrenergic, serotonergic)
Descending fibers from hypothalamus and brain stem to spinal cord



L3 Spinal Cord Cross Section

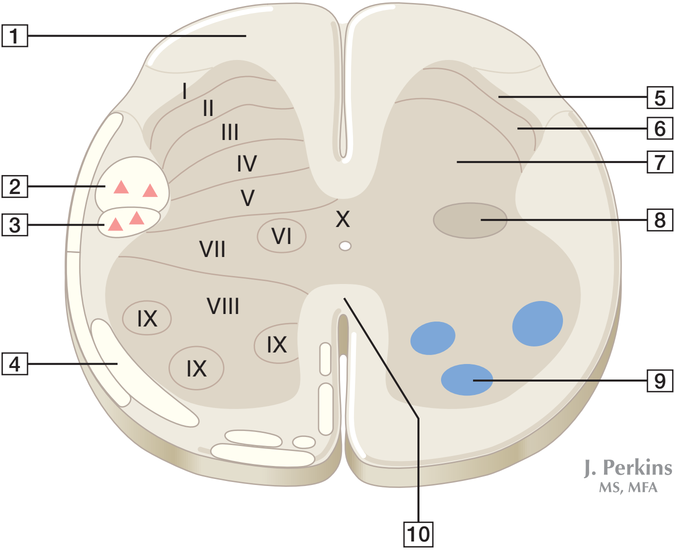
1. Fasciculus gracilis
2. Dorsolateral fasciculus (Lissauer's zone)
3. Dorsal spinocerebellar tract
4. Ventral spinocerebellar tract
5. Lateral (medullary) reticulospinal tract
6. Lateral vestibulospinal tract
7. Medial (pontine) reticulospinal tract
8. Anterior corticospinal tract
9. Medial longitudinal fasciculus
10. Anterior white commissure
11. Lower motor neurons in anterior horn
12. Nucleus dorsalis of Clarke

Comment: A spinal cord crush injury can damage local neurons at the site of the crush and disrupt both ascending and descending tracts. Such a lesion at a lumbar level would result in (1) flaccid paralysis of muscles supplied by the damaged levels (with loss of tone and reflexes); (2) spastic paralysis of muscles supplied by lower motor neurons below the level of the crush (with increased tone and muscle stretch reflexes, possible clonus, and pathological reflexes such as the plantar extensor response) due to loss of control from upper motor neurons; (3) loss of all sensation, epicritic (dorsal columns) and protopathic (anterolateral system) below the level of the lesion, although some protopathic sensation may remain; and (4) loss of bladder and bowel function, as well as sexual dysfunction due to loss of descending central fibers from the hypothalamus and brain stem. Initially the patient will likely experience spinal shock with flaccid muscle tone, flaccid bladder, and a paralyzed bowel, but within a week or so, both muscle tone and autonomic tone return and become exaggerated. Thus, the patient will experience a spastic bladder (small, reflexly stimulated to empty, with incontinence); loss of voluntary control over erectile function but with possible reflex erections; and increased gastrointestinal activity.

S3 Spinal Cord Cross Section

▲

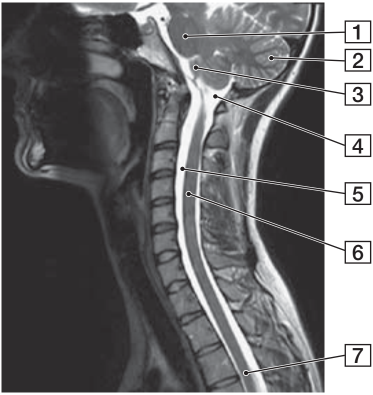
Descending monoamine axons
(noradrenergic, serotonergic)
Descending fibers from hypothalamus
and brain stem to spinal cord



S3 Spinal Cord Cross Section

1. Fasciculus gracilis
2. Lateral corticospinal tract
3. Rubrospinal tract
4. Anterolateral system
5. Marginal zone
6. Substantia gelatinosa
7. Nucleus proprius
8. Sacral parasympathetic nucleus (intermediomedial cell column)
9. Lower motor neurons in anterior horn
10. Anterior white commissure

Comment: In an adult, the sacral segments of the spinal cord are located at the level of the T1 vertebral body. Trauma or a tumor at this level can cause conus medullaris syndrome, which involves (1) flaccid paralysis of the muscles of the pelvic floor (lower motor neuron damage); (2) a flaccid bladder with overflow incontinence (loss of parasympathetic preganglionic neurons); (3) constipation and diminished bowel function (loss of parasympathetic preganglionic neurons); (4) loss of erectile function in males (loss of parasympathetic preganglionic neurons); (5) “saddle” anesthesia (loss of sensory processing); and (6) a possible sensation of pain experienced in the gluteal or perineal region.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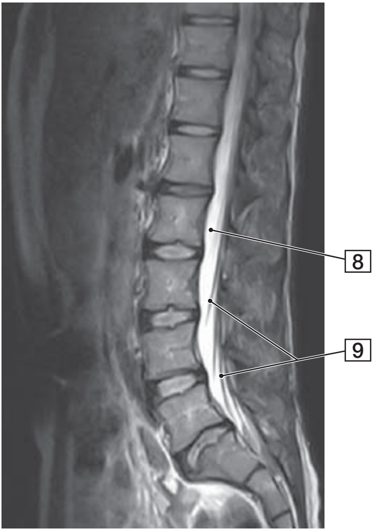
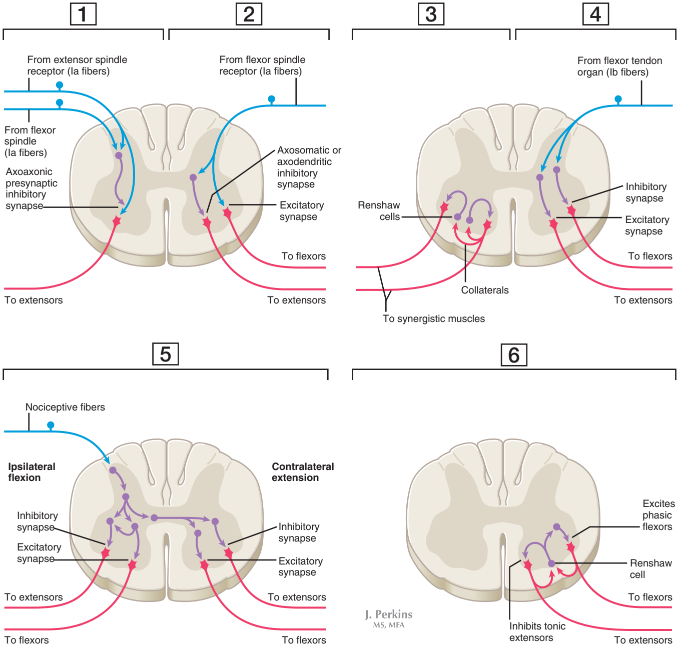

8
9

1. Pons
2. Cerebellum
3. Medulla
4. Cisterna magna
5. Subarachnoid space
6. Cervical spinal cord
7. Thoracic spinal cord
8. Lumbar cistern
9. Cauda equina—nerve roots in subarachnoid space

Comment: In an adult, the spinal cord ends at approximately the level of the L1 vertebral body. As a result of developmental growth of the spinal column relative to the spinal cord, the region below the L1 vertebral body consists of an enlarged subarachnoid space (the lumbar cistern) with cerebrospinal fluid, and passing nerve roots heading from their spinal cord segments to exit caudally at the appropriate intervertebral foramina. These nerve roots are not rigidly anchored, and have the ability to move slightly in the lumbar cistern; thus, a lumbar tap can be performed for withdrawal of cerebrospinal fluid without significant danger of damaging these passing nerve roots. However, a crush injury or severe trauma can sometimes damage these passing nerve roots in the lumbar cistern, producing cauda equina syndrome. This syndrome, in severe form, results in (1) flaccid bladder; (2) constipation and diminished bowel function; (3) loss of erectile function; (4) perineal anesthesia and additional sensory loss in the lower extremities; and (5) flaccid paralysis of muscles of the lower extremities. The pattern of sensory and motor loss in the lower extremities depends on the location of the injury and extent of damage to the passing nerve roots. This syndrome collectively consists of polyradiculopathies due to involvement of the nerve roots below the L1 vertebral level.

Spinal Somatic Reflex Pathways



Spinal Somatic Reflex Pathways

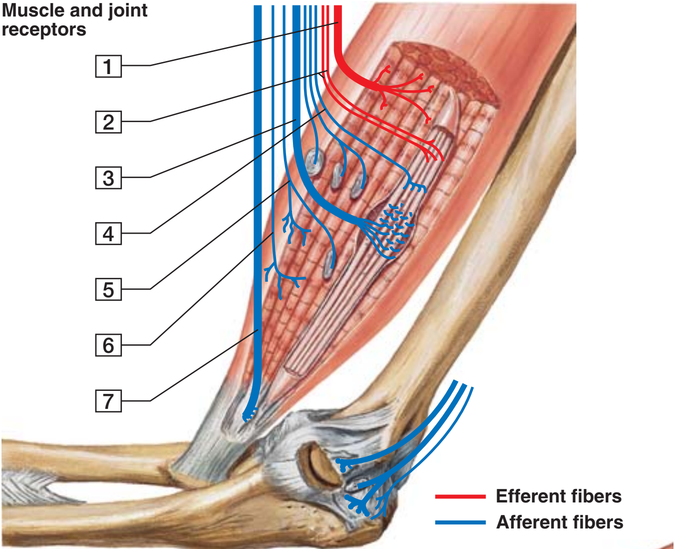
Type of Reflex Responses

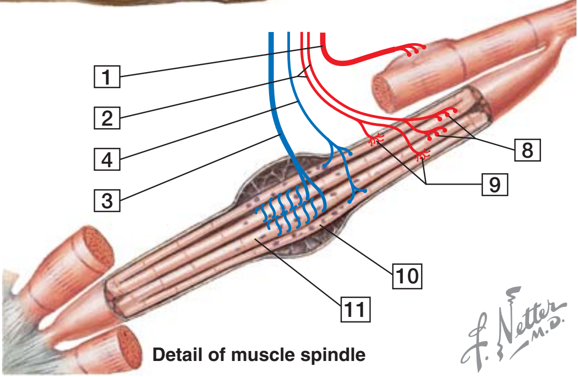
1. Afferent inhibition
2. Muscle stretch reflex
3. Recurrent inhibition
4. Golgi tendon organ reflex
5. Flexor reflex withdrawal reflex
6. Renshaw cell bias

Comment: Somatic spinal cord reflexes consist of a sensory input, a motor output, and possible intermediate neurons in the spinal cord. The muscle stretch reflex involves stimulation of Ia afferents from the muscle spindles responding to stretch of the homonymous muscle, synapsing on corresponding lower motor neurons (LMNs) to elicit contraction of the muscle whose tendon has been stretched. This reflex is the most commonly tested reflex in neurology. Ia afferents from flexor or extensor muscles can reciprocally inhibit the activity in their counterpart, called *reciprocal* or *afferent inhibition.* The Golgi tendon organ reflex is a disynaptic reflex stimulated by high threshold stretch of a tendon, leading to inhibition of the homonymous LMN pool to muscle and excitation of the antagonist LMN pool. This reflex is observable in patients with a pathological condition such as spasticity. The flexor reflex withdrawal response is a polysynaptic reflex elicited from sensory activation (particularly noxious stimuli) that results in appropriate reflex withdrawal of the body part from potential harm also accompanied by reciprocal inhibition. Renshaw cells provide a mechanism for controlling LMN excitability by inhibiting an LMN that has just been excited by a reflex activation or activation from a descending pathway. The Renshaw cell receives input from axon collaterals of both flexor and extensor LMNs, and in turn exerts an inhibitory bias toward extensor LMNs with a reciprocal excitation of flexor LMNs. This system favors flexor movements and helps to inhibit extensor movements, especially powerful antigravity muscle activity.

Muscle and Joint Receptors and Muscle Spindles

Muscle and joint receptors





Detail of muscle spindle

Muscle and Joint Receptors and Muscle Spindles

1. Alpha motor neuron fibers to extrafusal striated muscle end plates

2. Gamma motor neuron fibers in intrafusal striated muscle end plates

3. Ia (Aα) fibers from annulospiral endings (proprioceptive)

4. II (Aβ) fibers from flower spray endings (proprioceptors)

5. III (Aδ) fibers from free nerve endings and some specialized endings

6. IV (unmyelinated) fibers

7. Ib (Aα) fibers from Golgi tendon organs (proprioceptive)

8. γ1 plate endings

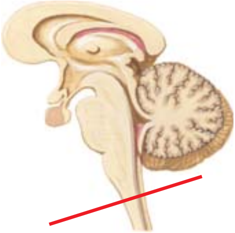
9. γ2 trail endings

10. Nuclear bag fiber

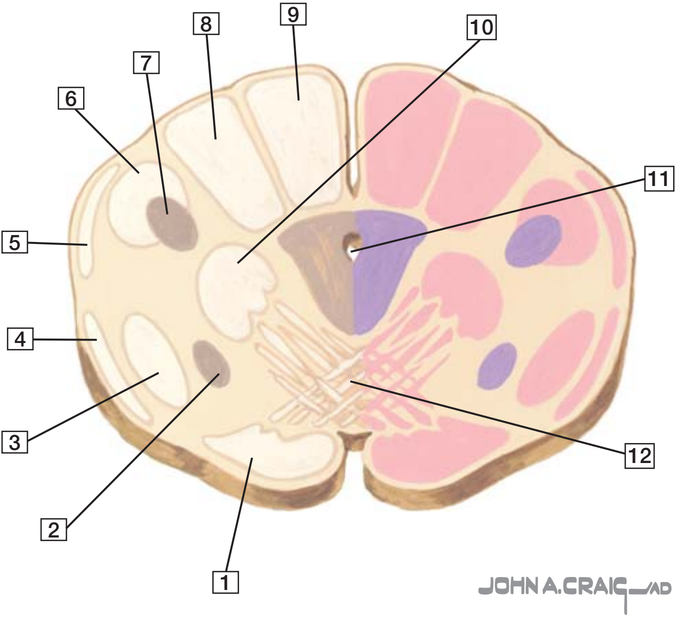
11. Nuclear chain fiber

Comment: Skeletal muscles are supplied by sensory and motor nerves and receptors. Sensory fibers are associated with the muscle spindle (sensory receptor) that lies in parallel with skeletal muscle fibers (extrafusal). Each spindle contains nuclear bag fibers (innervated by Ia afferents) and nuclear chain fibers (innervated by group II afferents). These afferents are responsive to tension on the muscle spindle, with group II reporting length information, and Ia reporting length and velocity information. Stretch of muscle (via tendon) results in Ia afferent activation, synapsing with homonymous LMNs monosynaptically, producing contraction in the affected muscle—muscle stretch reflex. Nuclear bag and chain fibers have contractile elements supplied by γ motor neurons from the spinal cord. These motor neurons cause the muscle spindle to stay in the dynamic range of excitability for all ranges of muscle activity, preventing muscle spindles from “unloading” and becoming unresponsive. In the absence of pathology, both α and γ motor neurons are coactivated by descending UMN systems. In pathological circumstances, the gamma system can be excessively activated, setting muscle spindles at a hair trigger, producing brisk (hyperreflexic) muscle stretch reflexes.

Brain Stem Cross Section: Medulla–Spinal Cord Transition



Level of section

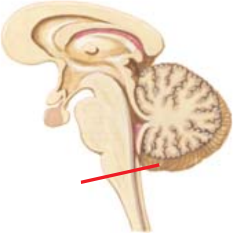


Brain Stem Cross Section: Medulla–Spinal Cord Transition

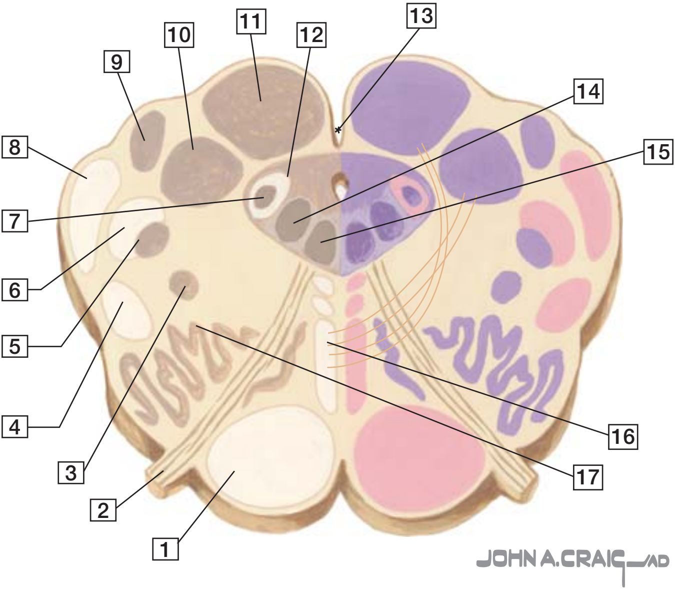
1. Pyramid
2. Nucleus CN XI
3. Spinothalamic/spinoreticular tracts (protopathic system)
4. Ventral spinocerebellar tract
5. Dorsal spinocerebellar tract
6. Spinal tract of CN V
7. Spinal nucleus of CN V
8. Fasciculus cuneatus
9. Fasciculus gracilis
10. Lateral corticospinal tract
11. Central canal
12. Decussation of the pyramids

Comment: This level is a transition between spinal cord and brain stem. Lower motor neurons (LMNs) cluster in neuronal groups in the ventral horn of the spinal cord; LMNs are in discrete nuclei within medial and lateral columns in the brain stem, the hypoglossal nucleus (CNN XII) and nucleus ambiguus. LMNs may be damaged in the brain stem in diseases such as bulbar polio and ALS, resulting in flaccid paralysis and wasting of muscles supplied (bulbar palsy). Damage to upper motor neurons (UMNs) supplying brain stem LMNs results in weakness or paralysis, but not wasting, of affected muscles. UMN disorder (pseudobulbar palsy) leaves LMNs physically intact. The descending UMN pathway from the cortex, found in the pyramid in the medulla, decussates (~80%) to form the lateral corticospinal tract (LCST) in the spinal cord. In the pyramid, damage to isolated cortical UMN fibers results in contralateral weakness and clumsiness of hand and finger movements, with mild tone loss. In the LCST, damage to UMN fibers in the spinal cord lateral funiculus involves damage to descending rubrospinal tract fibers, resulting in ipsilateral spastic paresis with hypertonus and hyperreflexia, classical UMN syndrome.

Brain Stem Cross Section: Medulla at the Level of the Obex



Level of section



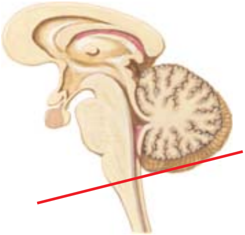
Brain Stem Cross Section: Medulla at the Level of the Obex

1. Pyramid
2. CN XII
3. Nucleus ambiguus
4. Spinothalamic/spinoreticular tracts (protopathic system)
5. Spinal nucleus of CN V
6. Spinal tract of CN V
7. Tractus solitarius
8. Inferior cerebellar peduncle
9. External (lateral) cuneate nucleus
10. Nucleus cuneatus
11. Nucleus gracilis
12. Nucleus solitarius
13. Obex
14. Dorsal motor nucleus of CN X
15. Nucleus of CN XII (hypoglossal)
16. Medial lemniscus
17. Inferior olivary nucleus

Comment: At this level, the primary sensory somatosensory nuclei, gracilis and cuneatus, are giving rise to the internal arcuate fibers, which cross the midline to form the medial lemniscus, found just dorsal to the medullary pyramids. At this point, the secondary sensory axons from both the epicritic system (medial lemniscus [ML]) and fast protopathic system (spinothalamic tract [STT]) are crossed, but are separated physically throughout the medulla, and can be separately involved in pathology (such as a paramedian stroke affecting the ML but not the STT). Damage results in contralateral loss of fine, discriminative touch; joint position sense; vibratory sensation (ML); or pain and temperature sensation (STT). Secondary sensory (and many higher order) axons carrying sensation for excruciating pain travel in the spino-reticular system and are represented bilaterally and more diffusely than the STT, accounting for the inability of isolated tractotomies to alleviate intractable pain permanently.

See book 11.3

Brain Stem Cross Section: Medulla at the Level of the Inferior Olive



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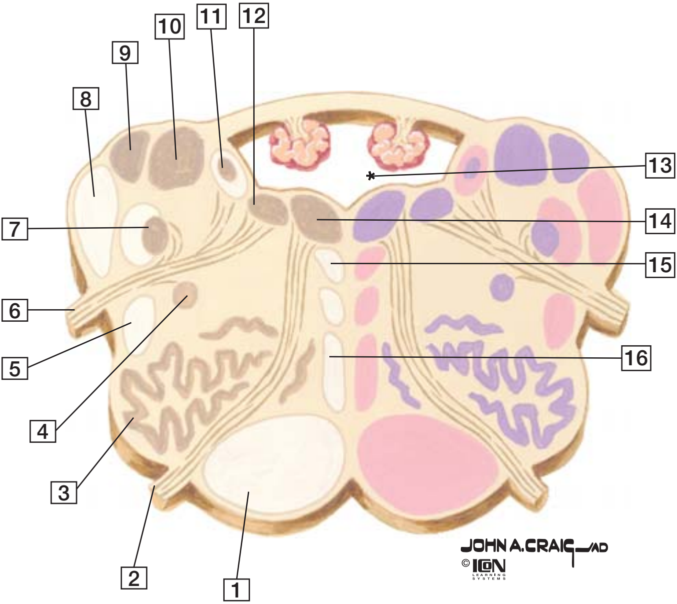

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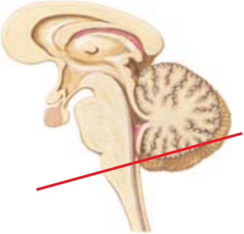
Brain Stem Cross Section: Medulla at the Level of the Inferior Olive

1. Pyramid
2. CN XII (hypoglossal)
3. Inferior olivary nucleus
4. Nucleus ambiguus
5. Spinothalamic/spinoreticular tracts (protopathic system)
6. CN X (vagus)
7. Spinal nucleus of CN V
8. Inferior cerebellar peduncle
9. External cuneate nucleus
10. Nucleus cuneatus
11. Nucleus solitaries
12. Dorsal motor nucleus of CN X
13. Fourth ventricle
14. Nucleus of CN XII
15. Medial longitudinal fasciculus
16. Medial lemniscus

Comment: The medulla is supplied with blood from paramedian and circumferential branches of the anterior spinal artery and vertebral artery. A paramedian vascular infarct can damage the pyramid (contralateral loss of skilled hand movements); medial lemniscus (contralateral loss of fine discriminative touch, joint position sense, vibratory sensation in the body); and ipsilateral cranial nerves (e.g., hypoglossal nerve, with paralysis of ipsilateral tongue muscles, resulting in protrusion to the contralateral side on command). A circumferential vascular infarct (posterior inferior cerebellar artery [PICA]) or vertebral artery infarct can damage the lateral field of the medulla (Wallenberg's syndrome or lateral medullary syndrome), resulting in (1) loss of pain and temperature sensation on the ipsilateral side of the face (descending nucleus and tract of V) and contralateral body (spinothalamic tract); (2) dysphagia and dysarthria (nucleus ambiguus); (3) ataxia of limbs and falling toward the ipsilateral side (inferior cerebellar peduncle); (4) vertigo with nausea and vomiting (vestibular nuclei); and ipsilateral Horner's syndrome (descending central autonomic axons).

Brain Stem Cross Section: Medulla at the Level of CN X and the Vestibular Nuclei



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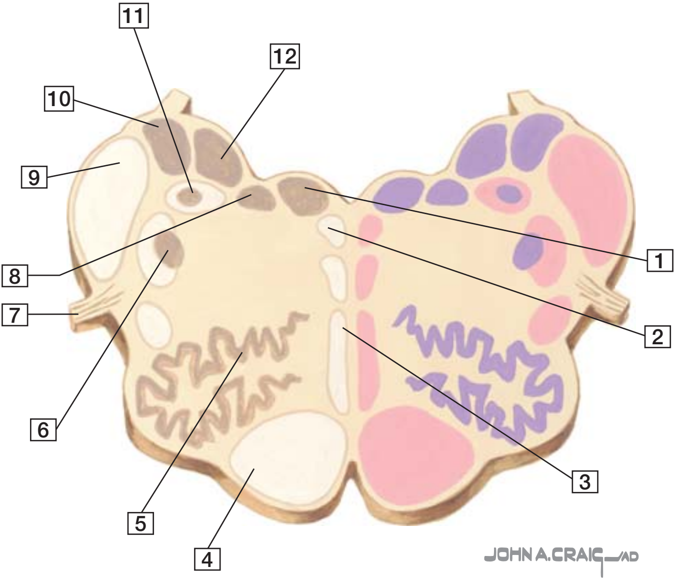
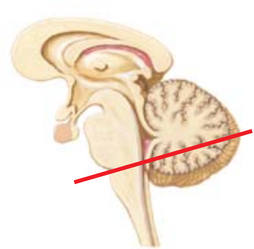


Brain Stem Cross Section: Medulla at the Level of CN X and the Vestibular Nuclei

1. Hypoglossal nucleus (CN XII)
2. Medial longitudinal fasciculus
3. Medial lemniscus
4. Pyramid
5. Inferior olivary nucleus
6. Spinal nucleus of CN V
7. CN X
8. Dorsal motor nucleus of CN X (vagus)
9. Inferior cerebellar peduncle
10. Inferior vestibular nucleus
11. Nucleus solitarius
12. Medial vestibular nucleus

Comment: In the medulla, the spinal tract (primary sensory axons) of CN V and its nucleus (secondary sensory) process pain and temperature sensation from the ipsilateral face and oral cavity. This system has representation of fast pain and temperature, as well as a component that connects into the reticular formation for prolonged, excruciating pain. In the medulla, damage to this system produces ipsilateral symptoms; above the medulla, secondary sensory decussation (trigeminothalamic tracts) carries that information to the contralateral side, with contralateral symptoms following damage to secondary sensory tracts. Sensory fibers from both the somatosensory system and trigeminal sensory system that convey proprioceptive information to the cerebellum travel through the ipsilateral inferior cerebellar peduncle as secondary sensory fibers into the cerebellar deep nuclei and cortex. The exception to this schema is the ventral spinocerebellar tract, which crosses through the anterior white commissure in the spinal cord, ascends through the inferior cerebellar peduncle, and crosses the midline a second time in the cerebellum. Because of the ipsilateral nature of these projections, damage to the cerebellum results in mainly ipsilateral symptoms (ataxia, dysmetria, dysdiadochokinesia, mild hypotonia).

Brain Stem Cross Section: Medullo-Pontine Junction



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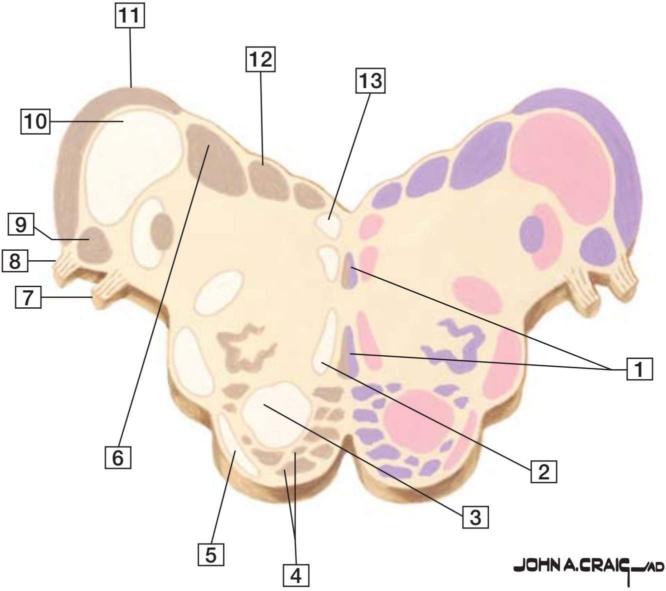
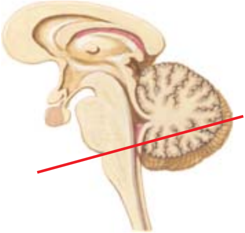


Brain Stem Cross Section: Medullo-Pontine Junction

1. Raphe nuclei (obscurus and pallidus)
2. Medial lemniscus
3. Corticospinal tract in basis pontis
4. Pontine nuclei
5. Middle cerebellar peduncle
6. Inferior vestibular nucleus
7. CN IX (glossopharyngeal)
8. CN VIII (vestibulocochlear)
9. Ventral cochlear nucleus
10. Inferior cerebellar peduncle
11. Dorsal cochlear nucleus
12. Medial vestibular nucleus
13. Medial longitudinal fasciculus

Comment: The dorsal and ventral cochlear nuclei (secondary sensory nuclei) receive auditory input from the spiral ganglion cells, which innervate hair cells on the basilar membrane. Cochlear nuclei project through acoustic striae to several brain stem auditory nuclei, including the superior olivary complex, lateral lemniscus nuclei, and inferior colliculus; these projections are both crossed and uncrossed and use the lateral lemniscus as a major ascending pathway. The recipient nuclei give rise to bilateral projections within the auditory system. A lesion at the level of the auditory nerve or cochlear nuclei can produce ipsilateral deafness or loss of hearing, but at the level of the brain stem and higher, ipsilateral lesions result in diminished hearing and contralateral neglect with simultaneous stimuli. The vestibular nuclei receive input from the vestibular (Scarpa's) ganglion, which innervates hair cells in the cristae ampullaris of the semicircular canals and in the maculae of the utricle and saccule. Vestibular nuclei convey information about linear acceleration (gravity) and angular acceleration (movement) to lower motor neurons for control of antigravity muscles and neck muscles, to extraocular muscles (for control of eye movements), and to the cerebellum (both primary and secondary axons) for coordination of tone and posture.

Brain Stem Cross Section: Pons at the Level of the Facial Nucleus



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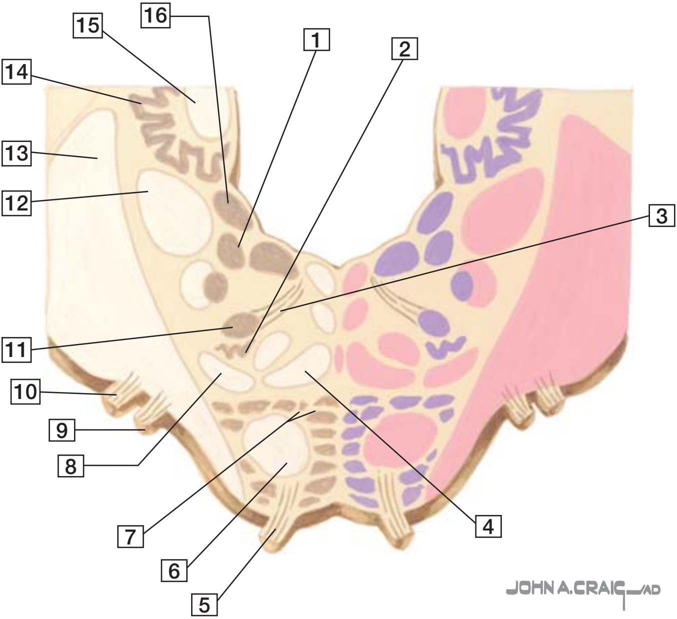
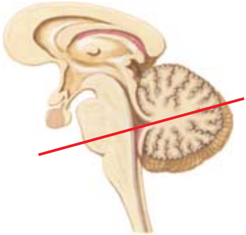


Brain Stem Cross Section: Pons at the Level of the Facial Nucleus

1. Lateral vestibular nucleus
2. Superior olivary nucleus
3. Fibers of CN VII
4. Medial lemniscus
5. CN VI (abducens)
6. Corticospinal tract
7. Trapezoid body
8. Spinothalamic/spinoreticular tracts (protopathic system)
9. CN VII (facial)
10. CN VIII (vestibulocochlear)
11. Nucleus of CN VII (facial)
12. Inferior cerebellar peduncle
13. Middle cerebellar peduncle
14. Dentate nucleus
15. Superior cerebellar peduncle
16. Superior vestibular nucleus

Comment: A vascular occlusion of a paramedian branch of the basilar artery in the lower pons results in medial inferior pontine syndrome. This syndrome includes (1) contralateral hemiplegia or hemiparesis (corticospinal system) and drooping of the lower face (corticobulbar tract); (2) contralateral loss of fine, discriminative touch, joint position sense, and vibratory sensation in the body, more severe in the upper extremity (medial lemniscus); (3) limb ataxia and gait apraxia (pontine nuclei and middle cerebellar peduncle crossing fibers bilaterally); (4) paralysis of lateral gaze in the ipsilateral eye and diplopia (CN VI); (5) paralysis of conjugate gaze to the ipsilateral side, with preservation of convergence (parapontine reticular formation); and (6) diplopia on attempted lateral gaze to the contralateral side (medial longitudinal fasciculus, internuclear ophthalmoplegia).

Brain Stem Cross Section: Pons at the Level of the Genu of the Facial Nerve



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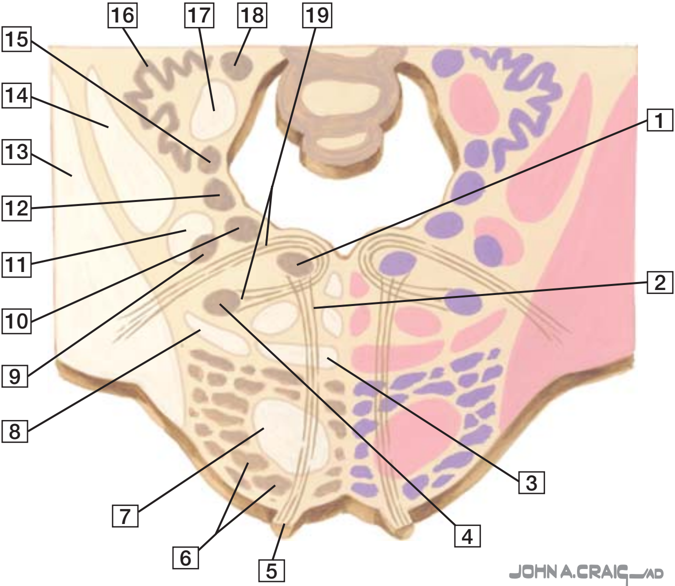
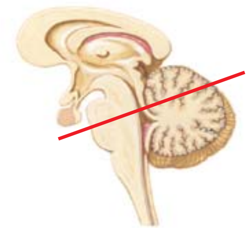


Brain Stem Cross Section: Pons at the Level of the Genu of the Facial Nerve

1. Nucleus of CN VI
2. Fibers of CN VI
3. Medial lemniscus
4. Nucleus of CN VII
5. CN VI (abducens)
6. Pontine nuclei
7. Corticospinal tract
8. Spinothalamic/spinoreticular tracts (protopathic system)
9. Spinal nucleus of CN V
10. Medial vestibular nucleus
11. Spinal tract of CN V (trigeminal)
12. Lateral vestibular nucleus
13. Middle cerebellar peduncle
14. Inferior cerebellar peduncle
15. Superior vestibular nucleus
16. Dentate nucleus
17. Superior cerebellar peduncle
18. Globose and emboliform nuclei
19. Fibers of CN VII (facial)

Comment: The pons is a frequent site of a hemorrhagic stroke, which is often large and lethal. A pontine hemorrhage can result in the rapid progression of total paralysis (quadriplegia), decerebrate posturing (upper motor neuron damage disinhibiting the lateral vestibular nuclei), coma, paralysis of ocular movements, and small but reactive pupils. A large infarct in the basilar artery may produce the same clinical picture. Some small lacunar infarcts may occur in the pons and may produce motor symptoms (corticospinal system) and/or ataxia (cerebellar peduncles, deep nuclei).

Brain Stem Cross Sections: Pons at the Level of the Trigeminal Motor and Main Sensory Nuclei



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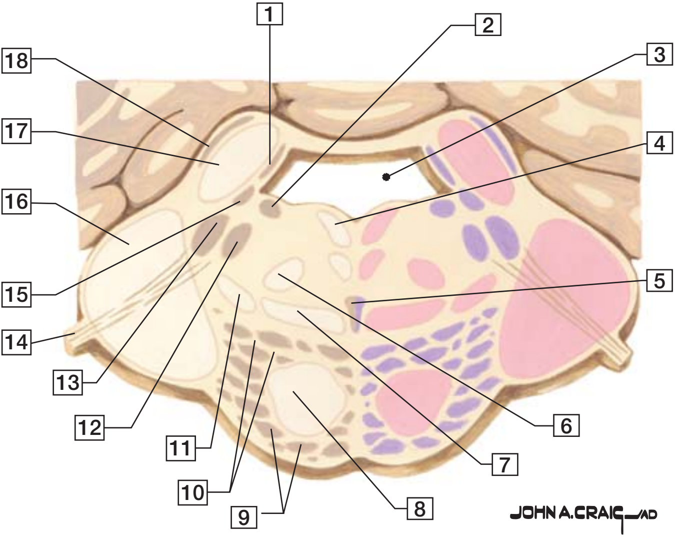
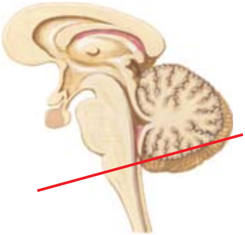


Brain Stem Cross Sections: Pons at the Level of the Trigeminal Motor and Main Sensory Nuclei

1. Medial parabrachial nucleus
2. Locus coeruleus
3. Fourth ventricle
4. Medial longitudinal fasciculus
5. Raphe nuclei (pontis)
6. Central tegmental tract
7. Medial lemniscus
8. Corticospinal tract
9. Pontine nuclei
10. Crossing fibers of the middle cerebellar peduncle
11. Spinothalamic/spinoreticular tracts (protopathic system)
12. Motor nucleus of V
13. Main (chief) sensory nucleus of V
14. CN V
15. Mesencephalic nucleus of V (trigeminal)
16. Middle cerebellar peduncle
17. Superior cerebellar peduncle
18. Lateral parabrachial nucleus

Comment: A vascular lesion of a circumferential branch of the basilar artery (anterior inferior cerebellar artery [AICA]) may produce lateral pontine syndrome, characterized by (1) contralateral loss of sensation in the body (both medial lemniscus and spinothalamic tract, which travel close together in the pons); (2) contralateral loss of pain and temperature sensation on the face (ventral trigeminal lemniscus, traveling on the dorsal surface of the medial lemniscus); (3) ipsilateral loss of epicritic sensation on the face (main sensory nucleus) or all general sensation on the face (CN V fibers); (4) ipsilateral lower motor neuron paralysis of muscles of mastication (motor nucleus of V); (5) limb ataxia (middle and superior cerebellar peduncles); (6) paralysis of conjugate gaze to the ipsilateral side (parapontine reticular formation); and (7) other pontine involvement (deafness, tinnitus, vertigo, nystagmus, facial palsy, Horner's syndrome).

Brain Stem Cross Section: Pons-Midbrain Junction



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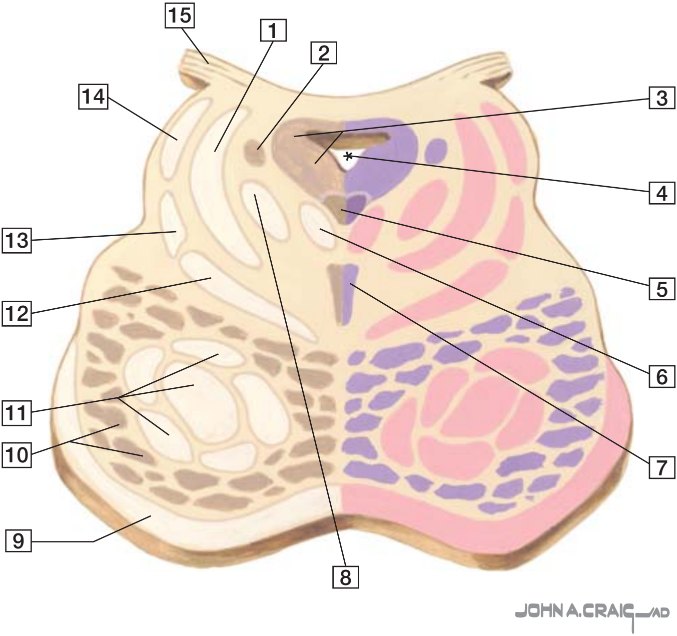


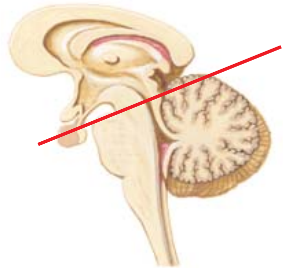
Brain Stem Cross Section: Pons-Midbrain Junction

1. Superior cerebellar peduncle
2. Locus coeruleus
3. Periaqueductal gray matter
4. Aqueduct
5. Dorsal raphe nucleus
6. Medial longitudinal fasciculus
7. Central superior raphe nucleus
8. Central tegmental tract
9. Middle cerebellar peduncle
10. Pontine nuclei
11. Corticospinal tract
12. Medial lemniscus
13. Spinothalamic/spinoreticular tracts (protopathic system)
14. Lateral lemniscus
15. CN IV (trochlear)

Comment: CNN IV is the only cranial nerve nucleus that sends its fibers contralaterally, innervating the superior oblique muscle; from a practical point of view, the nucleus is rarely lesioned and most damage occurs to the CN IV itself, resulting in ipsilateral inability to look down and in, resulting in difficulty reading in bed or when looking down, as well as negotiating curbs and stairs when walking. This brain stem region is also the site of major serotonergic (raphe dorsalis and central superior nuclei) and noradrenergic (locus coeruleus) nuclei, from which widespread projections emanate to virtually all major regions of the CNS and act as set-point regulators for other systems. These nuclei particularly influence affective behavior (target of antidepressive medication), visceral functions such as food intake and sexual behavior, pain processing and threshold, motor activation, and many other functions. The periaqueductal gray is an important zone of opioid peptide activity and nociceptive processing, particularly with chronic pain problems.

See book 11.10

Brain Stem Cross Section: Midbrain at the Level of the Inferior Colliculus



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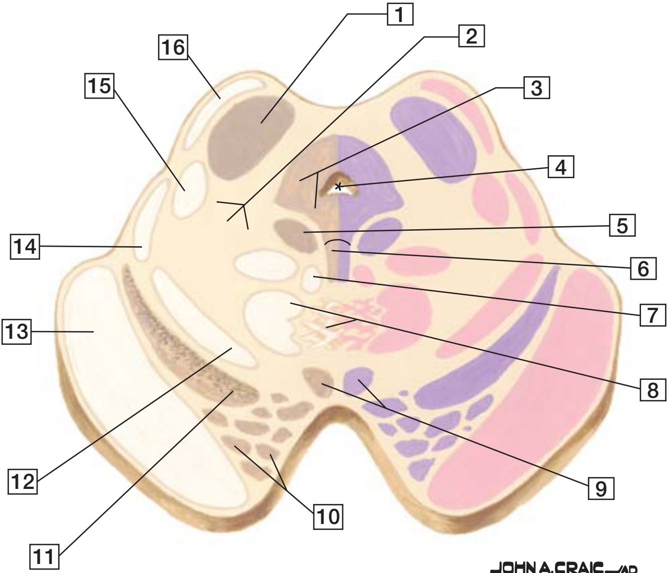
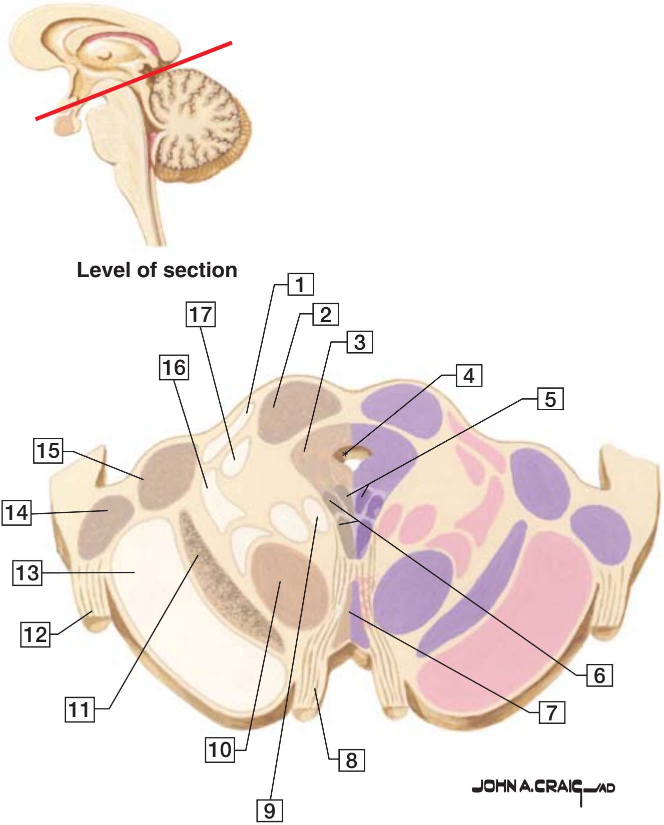


Brain Stem Cross Section: Midbrain at the Level of the Inferior Colliculus

1. Inferior colliculus
2. Reticular formation
3. Periaqueductal gray
4. Aqueduct
5. Nucleus of CN IV (trochlear)
6. Dorsal raphe nucleus
7. Medial longitudinal fasciculus
8. Superior cerebellar peduncle decussation
9. Interpeduncular nuclei
10. Pontine nuclei
11. Substantia nigra
12. Medial lemniscus
13. Cerebral peduncle
14. Spinothalamic/ spinoreticular tracts (protopathic system)
15. Lateral lemniscus
16. Brachium of the inferior colliculus

Comment: Space-occupying lesions in the forebrain, such as a hematoma (epidural or subdural), tumor, or increased intracranial pressure can force herniation of the forebrain through the tentorium cerebelli. Such a herniation displaces the thalamus and midbrain downward, removing functional capabilities of the rostral structures affected. Major resultant problems include (1) progressive loss of consciousness, from drowsiness to stupor to coma (consciousness requires an intact reticular formation and one functioning hemisphere); (2) decerebrate state (extensor posturing of all four limbs) from corticospinal and rubrospinal tract impairment that disinhibits the lateral vestibulospinal system, accompanied by opisthotonus (neck extended) and plantar extensor responses; (3) breathing changes, progressing from Cheyne-Stokes (crescendo-decrescendo) with diencephalic-midbrain damage, to shallow or ataxic breathing with further brain stem involvement; (4) loss of pupillary light reflexes from compression of CN III against the free edge of the tentorium; and (5) loss of vertical and conjugate eye movements from damage to the visual tectum.

Brain Stem Cross Section: Midbrain at the Level of the Superior Colliculus and Geniculate Nuclei

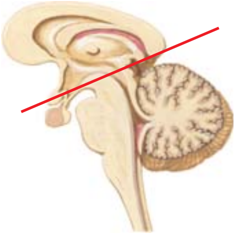


Brain Stem Cross Section: Midbrain at the Level of the Superior Colliculus and Geniculate Nuclei

1. Brachium of the inferior colliculus
2. Superior colliculus
3. Periaqueductal gray
4. Aqueduct
5. Nucleus of Edinger-Westphal (III)
6. Nucleus of CN III (oculomotor)
7. Ventral tegmental area
8. CN III
9. Medial longitudinal fasciculus
10. Red nucleus
11. Substantia nigra
12. Optic tract
13. Cerebral peduncle
14. Lateral geniculate nucleus (body)
15. Medial geniculate nucleus (body)
16. Medial lemniscus
17. Spinothalamic/spinoreticular tracts (protopathic system)

Comment: A vascular lesion of the paramedian blood supply to the upper midbrain (from branches of the posterior cerebral or posterior communicating arteries) results in Weber's syndrome, characterized by contralateral hemiplegia (cerebral peduncle), contralateral drooping lower face (corticobulbar tract in the cerebral peduncle), lateral strabismus and extraocular paralysis ipsilaterally (CN III motor component), and a fixed, dilated pupil ipsilaterally (CN III parasympathetic component). Vascular damage involving more distal paramedian branches in the upper midbrain (Benedict's syndrome) may include more extensive structural damage to the red nucleus, substantia nigra, pallidothalamic fibers, and dentatorubral and dentatothalamic fibers; such damage may be accompanied by akinesia, intention tremor, or choreoathetoid movements.

Brain Stem Cross Section: Midbrain-Diencephalic Junction



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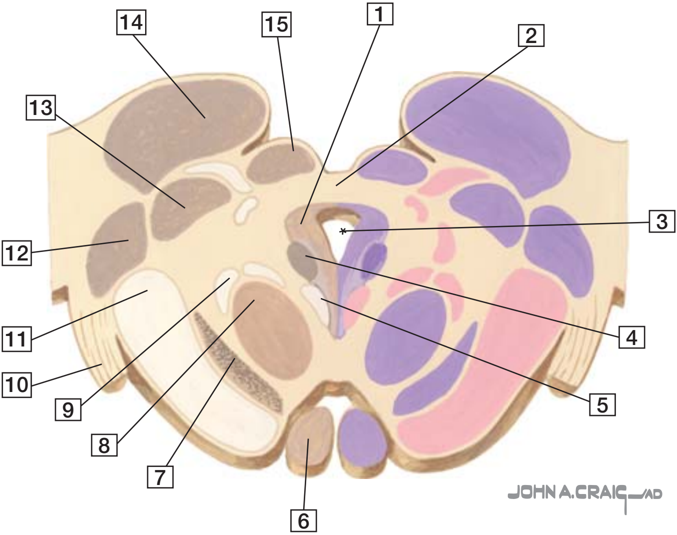


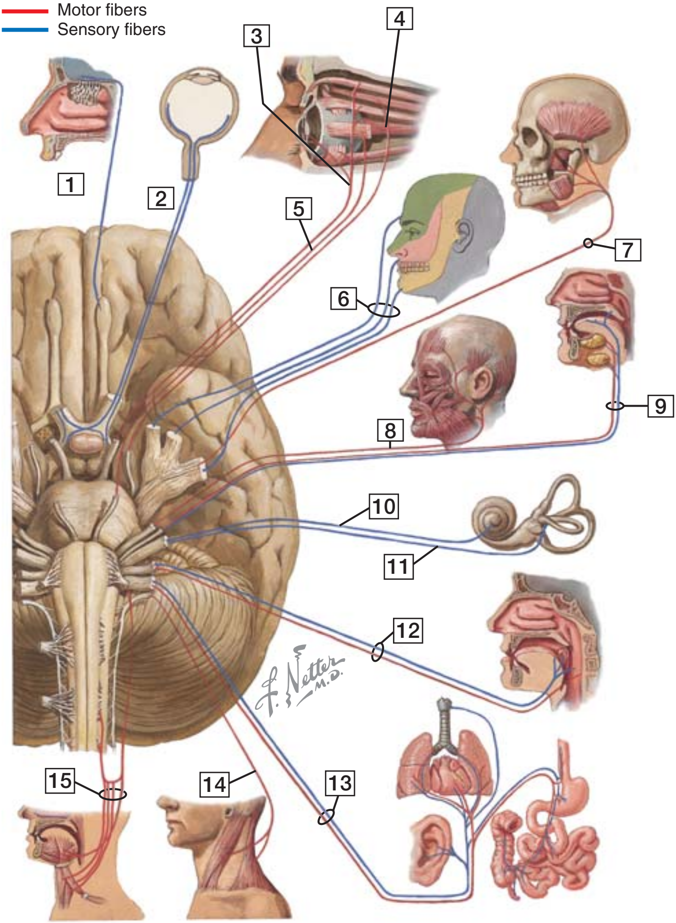
Brain Stem Cross Section: Midbrain-Diencephalic Junction

1. Periaqueductal gray
2. Posterior commissure
3. Aqueduct
4. Nucleus of Darkschewitsch
5. Medial longitudinal fasciculus
6. Mammillary bodies (nuclei)
7. Substantia nigra
8. Red nucleus
9. Cerebellorubrothalamic tract (fibers)
10. Optic tract
11. Cerebral peduncle
12. Lateral geniculate nucleus (body)
13. Medial geniculate nucleus (body)
14. Pulvinar
15. Superior colliculus

Comment: At this level of transition between the midbrain and diencephalon, the optic tract sends secondary sensory axons to the superior colliculus, the pretectum, the lateral geniculate nucleus, the hypothalamus (suprachiasmatic nucleus), and nucleus of the accessory optic tract. The lateral geniculate nucleus provides processing for the main visual interpretation of the visual world through the geniculo-calcarine pathway. The superior colliculus provides a second visual pathway for localizing information through the associative visual cortex. The pretectum mediates the pupillary light reflex, with the crossed component traveling via the posterior commissure. The suprachiasmatic nucleus of the hypothalamus helps to regulate diurnal cycles and circadian rhythms. The nucleus of the accessory optic tract may assist brain stem responses for visual tracking and help to mediate visual connections to the preganglionic sympathetic neurons in T1 and T2 connecting to the superior cervical ganglion.

See book 11.14

Cranial Nerves: Basal View of the Brain

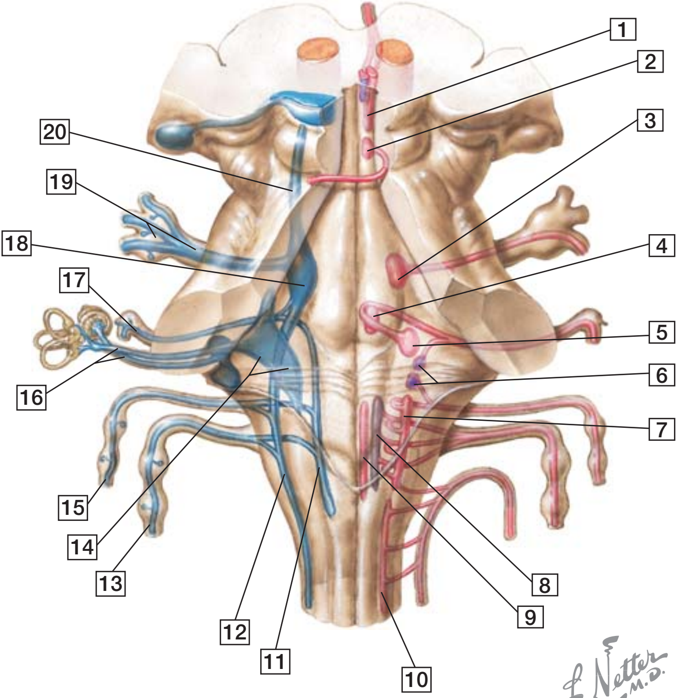


Cranial Nerves: Basal View of the Brain

1. CN I Olfactory
2. CN II Optic
3. CN III Oculomotor
4. CN IV Trochlear
5. CN VI Abducens
6. CN V Trigeminal
7. Motor component of CN V (Mandibular division)
8. CN VII Facial
9. Nervus intermedius
10. Cochlear division of CN VIII Vestibulocochlear
11. Vestibular division of CN VIII Vestibulocochlear
12. CN IX Glossopharyngeal
13. CN X Vagus
14. CN XI Spinal Accessory
15. CN XII Hypoglossal

Comment: CNs I and II are central nervous system tracts that are myelinated by oligodendroglia. The optic nerve is surrounded by meninges and a subarachnoid space, allowing conveyance of increased intracranial pressure, resulting in papilledema. CNs III–XII are peripheral nerves when they exit the brain stem (central portion of axons myelinated by oligodendroglia, peripheral part of axons myelinated by Schwann cells). These cranial nerves contain sensory, motor, and autonomic components; peripheral damage or demyelination involves these three functional components, while central damage or demyelination may involve additional pathology to central tracts. Some types of pathology may affect multiple cranial nerves, such as aneurysm or thrombosis in the cavernous sinus (CNs III, IV, VI, V1); a tumor in the middle cranial fossa in the retrosphenoid space (CNs III–VI); an acoustic schwannoma at the cerebellopontine angle (CNs VII and VIII, sometimes expanding to include CNs V and IX); tumors and aneurysms of the jugular foramen (CNS IX, X, and XI); or granulomatous lesions in the posterior retroparotid space (CNs IX–XII).

Cranial Nerves and Their Nuclei: Schematic View



🟥 Efferent fibers and nuclei—motor

🟦 Afferent fibers and nuclei

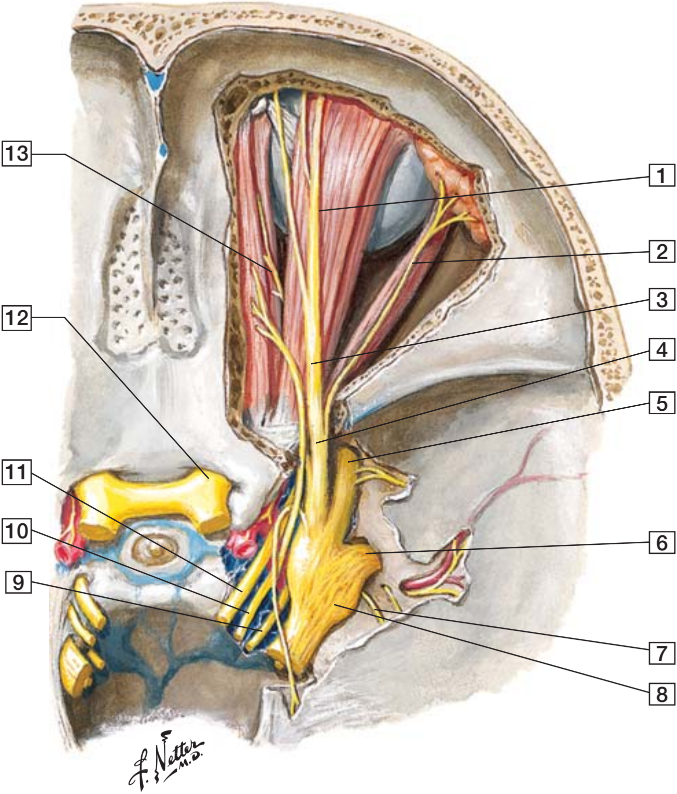
🟪 Efferent fibers and nuclei—autonomic

Cranial Nerves and Their Nuclei: Schematic View

1. Oculomotor nucleus
2. Trochlear nucleus
3. Motor nucleus of CN V
4. Abducens nucleus
5. Facial motor nucleus
6. Superior and inferior salivatory nuclei
7. Nucleus ambiguus
8. Dorsal motor nucleus of CN X
9. Hypoglossal nucleus
10. Nucleus of CN XI (spinal accessory)
11. Nucleus solitarius
12. Spinal tract and spinal nucleus of CN V
13. CN X (vagus)
14. Vestibular nuclei
15. CN IX (glossopharyngeal)
16. CN VIII (vestibulocochlear)
17. CN VII (facial)
18. Principal (chief, main) sensory nucleus of CN V
19. CN V (trigeminal) and ganglion
20. Mesencephalic nucleus of CN V

Comment: CNs I and II are central nervous system tracts. CNs V and VII–X have primary afferent components that terminate in associated secondary sensory nuclei (trigeminal descending and main sensory nuclei, nucleus solitarius, cochlear nuclei, vestibular nuclei). Some primary sensory tracts of CNs are present longitudinally in brain stem (descending tract of V, tractus solitarius, mesencephalic tract of V). Motor cranial nerve nuclei are found in two longitudinal columns, a medial column (CNs III, IV, VI, XII) and a lateral column (CNs V and VII [motor] and nucleus ambiguus). These are classic lower motor neurons with direct connections with skeletal muscle fibers ending as neuromuscular junctions. Specialized nuclei for parasympathetic preganglionic neurons, including nucleus of Edinger-Westphal (III), superior salivatory nucleus (VII), inferior salivatory nucleus (IX), and dorsal motor nucleus of CN X, connect with parasympathetic ganglia in the periphery near their target effector tissue.

See book 11.16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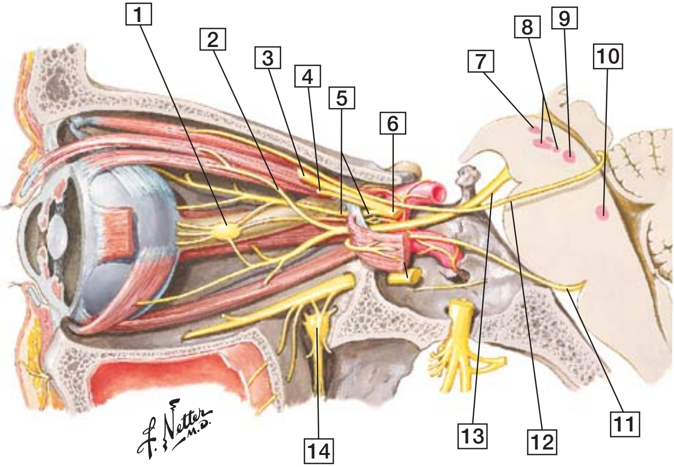
F. Netter M.D.

Nerves of the Orbit

1. Supraorbital nerve
2. Lacrimal nerve
3. Frontal nerve
4. Ophthalmic nerve (CN V1)
5. Maxillary nerve (CN V2)
6. Mandibular nerve (CN V3)
7. Greater petrosal nerve
8. Trigeminal (semilunar) ganglion
9. Abducens nerve (CN VI)
10. Trochlear nerve (CN IV)
11. Oculomotor nerve (CN III)
12. Optic nerve (CN II)
13. Nasociliary nerve

Comment: CN II is a central tract carrying visual information to further central sites from the ipsilateral retina. CNs III (motor component), IV, and VI are lower motor neurons that innervate extraocular muscles. Sensory fibers of CN V1 supply general sensation to the cornea and eyeball and provide the afferent limb of the corneal reflex. Motor fibers of CN VII innervate the orbicularis oculi muscles, closing the eye; these fibers provide the efferent limb of the corneal reflex. CN II may be damaged by a central demyelinating disease (multiple sclerosis), an optic nerve glioma, ischemia (central retinal artery), or injury (sphenoid fracture). The resultant deficit is ipsilateral blindness or a scotoma (blind spot) corresponding to the site of the damaged fibers; the ipsilateral nature of the deficit rules out optic chiasm or optic tract involvement. The retina is CNS tissue and may undergo macular degeneration, leading to an inability to read and loss of acuity. The retina also may be affected by increased intracranial pressure conveyed through the CSF in the subarachnoid space surrounding the optic nerve.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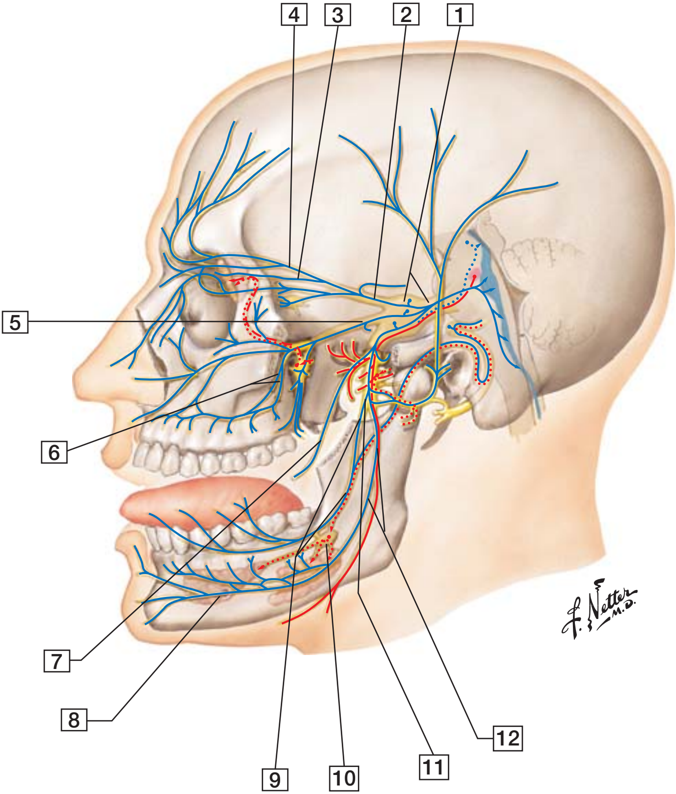
Extraocular Cranial Nerves

1. Ciliary ganglion
2. Superior division of CN III
3. Frontal nerve
4. Lacrimal nerve
5. Nasociliary nerve
6. Ophthalmic nerve (CN V1)
7. Nucleus of Edinger-Westphal (CN III parasympathetic component)
8. Oculomotor nucleus
9. Trochlear nucleus
10. Abducens nucleus
11. Abducens nerve (CN VI)
12. Trochlear nerve (CN IV)
13. Oculomotor nerve (CN III)
14. Pterygopalatine ganglion

Comment: CNN III (oculomotor nucleus) innervates medial rectus, superior rectus, inferior rectus, and inferior oblique muscles; damage results in lateral strabismus, inability to adduct the ipsilateral eye, and diplopia. This nucleus innervates the levator palpebrae superioris muscle; damage results in profound ptosis of the ipsilateral eye. A lesion of CN III damages preganglionic parasympathetic outflow from the nucleus of Edinger-Westphal, resulting in a fixed, dilated ipsilateral pupil. CN IV innervates the superior oblique muscle; damage results in an inability to look downward when looking nasally. The patient has difficulty walking down stairs or off curbs and diplopia when trying to read laying down; compensates by avoiding use of the denervated superior oblique and turns head away from the side of the lesion. CN VI innervates the lateral rectus muscle; damage results in medial strabismus, inability to move the ipsilateral eye laterally, and diplopia on attempted lateral gaze. The ciliary ganglion gives rise to postganglionic parasympathetic axons that supply the pupillary constrictor muscle and the ciliary muscle; damage results in a fixed, dilated pupil that does not constrict for the pupillary light reflex and does not accommodate to near vision.

Trigeminal Nerve (CN V)



━ Motor fibers

━ Sensory fibers

• • • • Proprioceptive fibers

• • • • Parasympathetic fibers

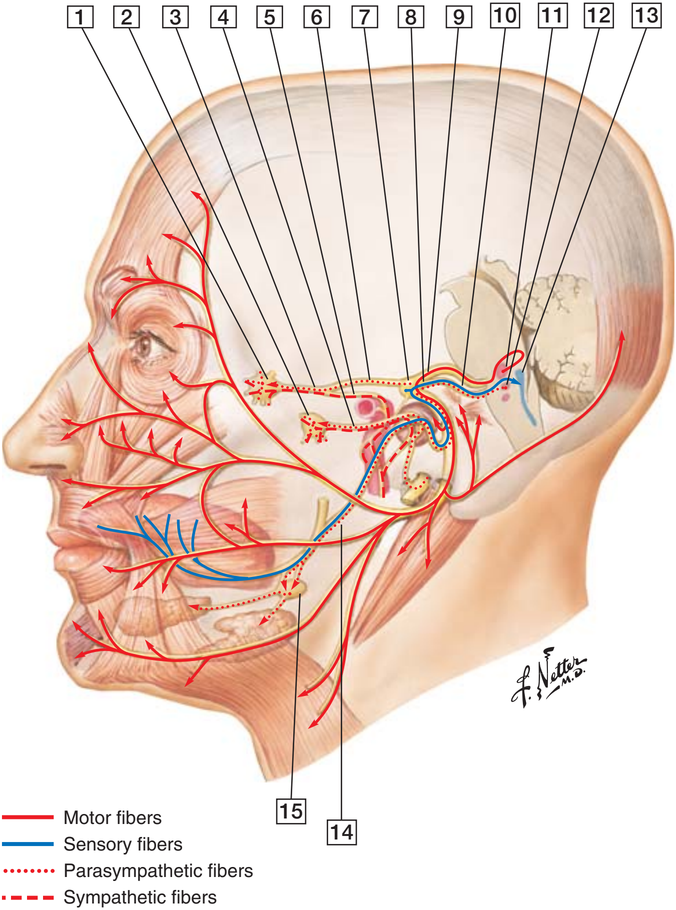
▬ ▬ ▬ Sympathetic fibers

Trigeminal Nerve (CN V)

1. Trigeminal nerve (CN V)
2. Ophthalmic nerve (CN V1)
3. Lacrimal nerve
4. Frontal nerve
5. Maxillary nerve (CN V2)
6. Superior alveolar branches of infraorbital nerve
7. Buccal nerve
8. Mental nerve
9. Lingual nerve
10. Submandibular ganglion
11. Mandibular nerve (CN V3)
12. Inferior alveolar nerve

Comment: The trigeminal nerve carries sensory information from the face, sinuses, teeth, and anterior portion of the oral cavity. Its three subdivisions, the ophthalmic (V1), maxillary (V2), and mandibular (V3) nerves, have distinct boundaries and do not overlap in innervation, damage to a division of CN V results in total anesthesia in the territory of innervation. The mandibular nerve also carries motor fibers from the motor nucleus to the muscles of mastication and the tensor tympani muscle. The trigeminal ganglion central axons terminate in the descending nucleus of V (lower two thirds process pain and temperature from the ipsilateral face) and the main (chief) sensory nucleus (upper portion of descending nucleus of V and main sensory nucleus process fine, discriminative touch). The mesencephalic nucleus of V is a primary sensory nucleus in the CNS (the only example) with peripheral axons innervating muscle spindles in the muscles of mastication and the extraocular muscles. The trigeminal nerve subdivisions are subject to a disorder, trigeminal neuralgia (tic douloureux), consisting of sudden, brief, excruciating paroxysms of stabbing or lancinating pain in the territory of that branch. There is often a trigger point that sets off a paroxysm.

Facial Nerve (CN VII)

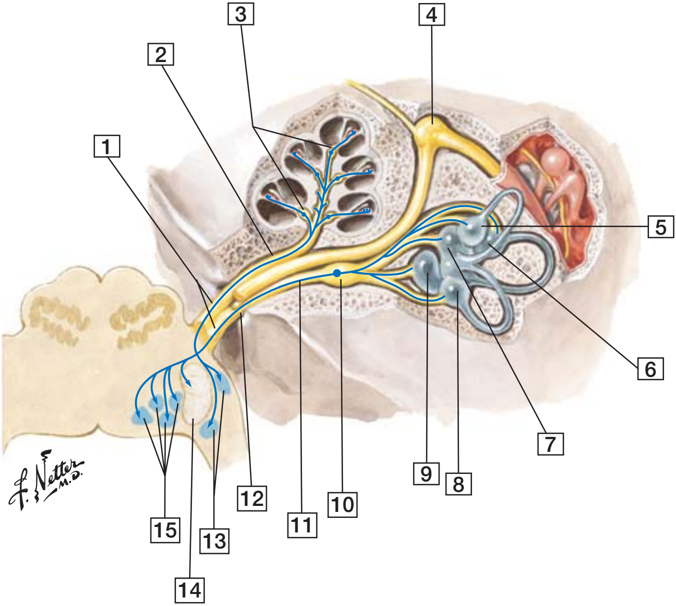


Facial Nerve (CN VII)

1. Pterygopalatine ganglion
2. Otic ganglion
3. Nerve of the pterygoid canal
4. Lesser petrosal nerve
5. Deep petrosal nerve
6. Greater petrosal nerve
7. Geniculate ganglion
8. Facial nerve (CN VII)
9. Motor root of CN VII
10. Nervus intermedius of CN VII
11. Motor nucleus of CN VII
12. Superior salivatory nucleus
13. Nucleus of the solitary tract (nucleus solitarius)
14. Chorda tympani
15. Submandibular ganglion

Comment: CN VII has sensory, motor, and autonomic components. Motor fibers of the facial nerve motor nucleus innervate the muscles of facial expression; damage results in ipsilateral paralysis of facial expression, including the forehead (Bell's palsy); lesions of central corticobulbar fibers result in drooping of the lower face, with no forehead involvement. The facial nucleus also innervates the stapedius muscle, which dampens the ossicles in the presence of loud noise; damage results in hyperacusis. Parasympathetic preganglionic fibers from the superior salivatory nucleus innervate the pterygopalatine ganglion (innervates lacrimal glands) and the submandibular ganglion (innervates submandibular and sublingual salivary glands); damage results in ipsilateral diminished tearing and salivation. Special sensory taste fibers from the anterior two thirds of the tongue (via chorda tympani) and the soft palate (via greater petrosal nerve), whose cell bodies are found in the geniculate ganglion, convey taste information to the rostral portion of nucleus solitarius in the medulla; taste remains mainly ipsilateral in its course of central processing.

Vestibulocochlear Nerve (CN VIII)

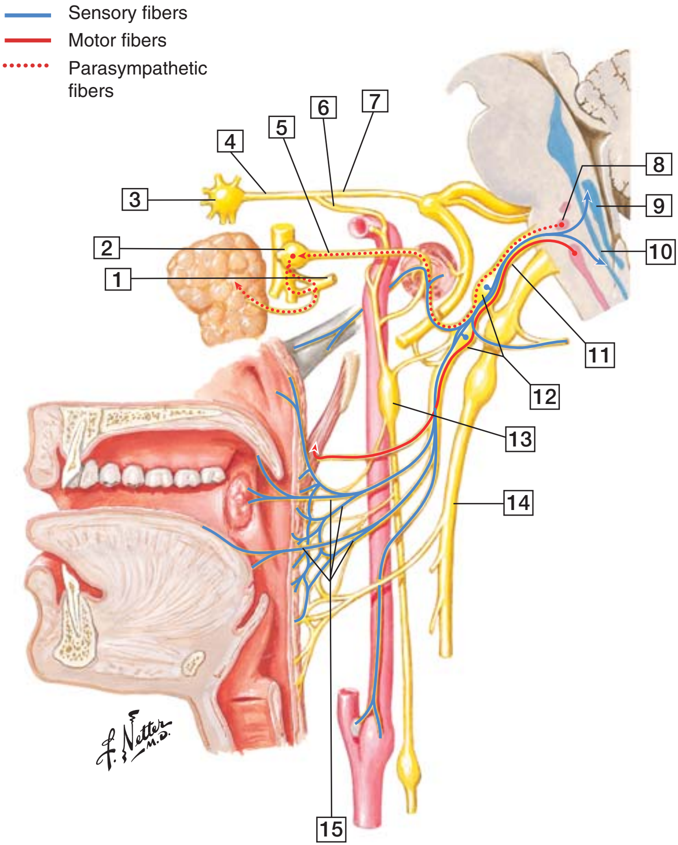


Vestibulocochlear Nerve (CN VIII)

1. Vestibulocochlear nerve (CN VIII)
2. Cochlear part of CN VIII
3. Spiral ganglion
4. Geniculate ganglion of CN VII
5. Ampulla of superior semicircular duct
6. Ampulla of lateral semicircular duct
7. Utricle
8. Ampulla of posterior semicircular duct
9. Saccule
10. Vestibular ganglion (Scarpa's ganglion)
11. Vestibular part of CN VIII
12. Internal acoustic meatus
13. Cochlear nuclei
14. Inferior cerebellar peduncle
15. Vestibular nuclei

Comment: Vestibular division of CN VIII arises from bipolar primary sensory neurons in the vestibular (Scarpa's) ganglion. The peripheral process innervates hair cells in the utricle and saccule that respond to linear acceleration (gravity) and in the ampullae of the semicircular ducts that respond to angular acceleration (movement); the central process of ganglion cells terminates in the vestibular nuclei and vestibular portion of the cerebellum. Vestibular nerve irritation (e.g., acoustic schwannoma) can result in vertigo, dizziness, nausea, and unsteadiness of spatial disorientation. These symptoms persist with nerve destruction. Auditory division of CN VIII arises from bipolar primary sensory neurons in spiral ganglion. The peripheral process innervates hair cells that lie along the organ of Corti on the basilar membrane in the cochlear duct. The central process of ganglion cells conveys hearing information to the cochlear nuclei. Auditory nerve irritation may first produce tinnitus (ringing, roaring), followed by slow loss of hearing and inability to determine the direction from which a sound is coming. Destruction of the auditory nerve results in profound ipsilateral deafness.

Glossopharyngeal Nerve (CN IX)

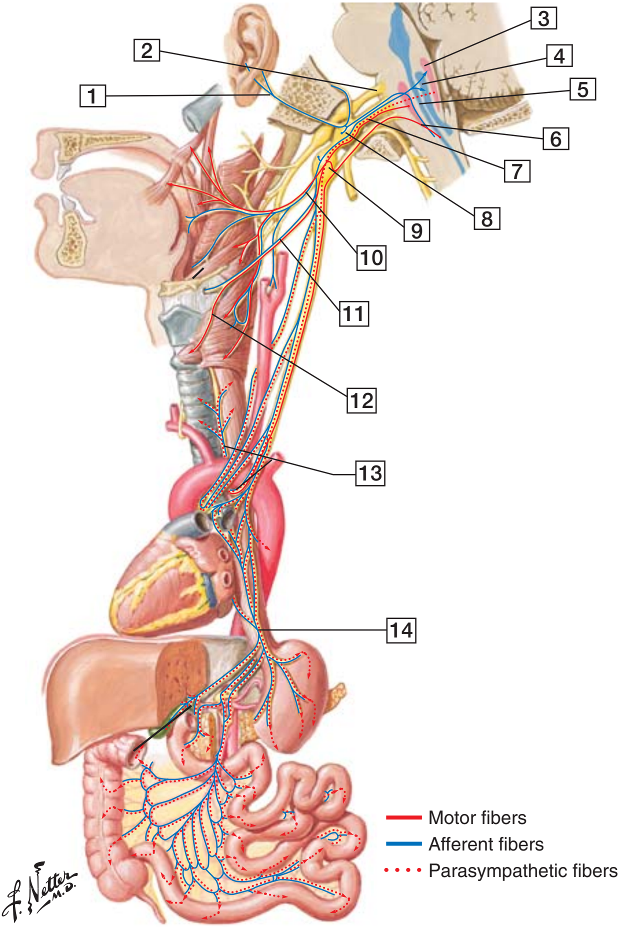


Glossopharyngeal Nerve (CN IX)

1. Auriculotemporal nerve
2. Otic ganglion
3. Pterygopalatine ganglion
4. Nerve of the pterygoid canal
5. Lesser petrosal nerve
6. Deep petrosal nerve
7. Greater petrosal nerve
8. Inferior salivatory nucleus
9. Nucleus solitarius
10. Spinal tract and descending (spinal) nucleus of CN V
11. Glossopharyngeal nerve (CN IX)
12. Superior and inferior ganglia of CN IX
13. Superior cervical ganglion (sympathetic)
14. Vagus nerve (CN X)
15. Pharyngeal, tonsillar, and lingual branches of CN IX

Comment: CN IX has sensory, motor, and autonomic components. Motor fibers from nucleus ambiguus supply the stylopharyngeus muscle and may assist in innervation of pharyngeal muscles for swallowing. Preganglionic parasympathetic axons from the inferior salivatory nucleus travel with CN IX to the otic ganglion, which supplies the parotid salivary gland. Special sensory axons from the petrosal (inferior) ganglion of IX carry information from taste buds on the posterior one third of the tongue and part of the soft palate and terminate in the rostral portion of nucleus solitarius. Additional primary sensory axons in the inferior ganglion of IX convey general sensation from the posterior third of the tongue and from the pharynx, fauces, tonsils, tympanic cavity, eustachian tube, and mastoid cells; the central portion of the axon terminates in the descending nucleus of V. General sensory fibers provide the afferent limb of the gag reflex. Additional primary sensory neurons innervate the carotid body (CO, chemoreception) and carotid sinus (baroceptors) and convey this information to the caudal nucleus solitarius. Primary sensory axons from the superior ganglion of IX innervate a small region behind the ear and convey general sensation into the descending nucleus of V. The glossopharyngeal nerve can be subject to brief, excruciating paroxysms of pain (glossopharyngeal neuralgia), similar to trigeminal neuralgia.

Vagus Nerve (CN X)

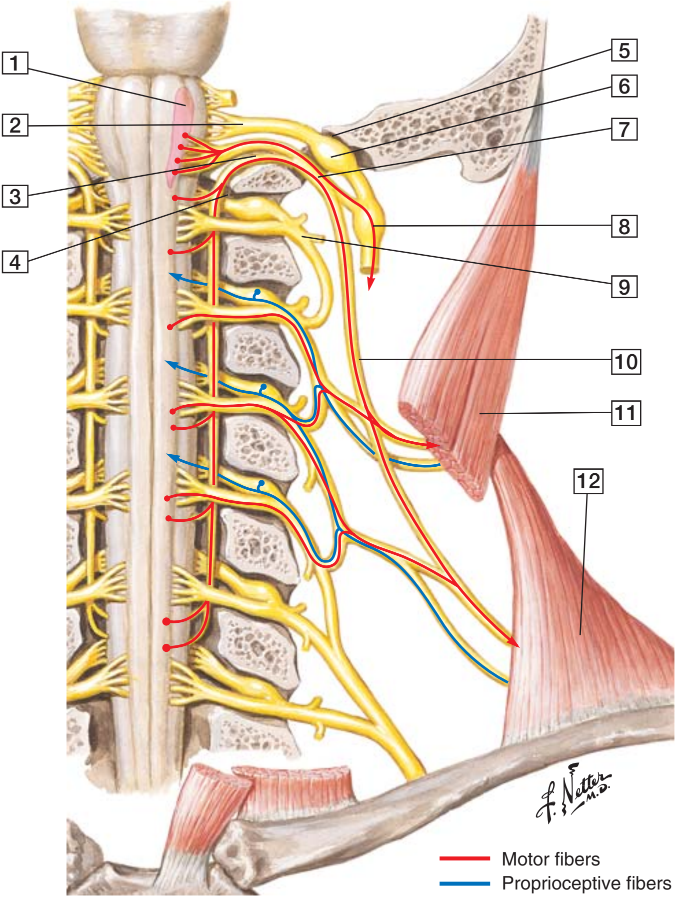


Vagus Nerve (CN X)

1. Auricular branch of CN X
2. Glossopharyngeal nerve (CN IX)
3. Dorsal motor nucleus of CN X
4. Nucleus solitarius
5. Spinal (descending) tract and nucleus of CN V
6. Nucleus ambiguus
7. Vagus nerve (CN X)
8. Superior ganglion of CN X
9. Inferior ganglion of CN X
10. Pharyngeal branch of CN X
11. Vagal branch to carotid sinus
12. Superior laryngeal nerve
13. Left recurrent laryngeal nerve
14. Anterior vagal trunk

Comment: CN X has sensory, motor, and autonomic components. Lower motor neurons from nucleus ambiguus travel with CN X to supply muscles of the soft palate, pharynx, and larynx; they control speaking and swallowing. A unilateral lesion of CN X results in drooping of the soft palate, hoarseness and nasal speech, dysphagia, and decreased gag reflex. Bilateral nerve damage results in loss of speech and swallowing. Preganglionic neurons from the dorsal motor nucleus of X distribute to intramural ganglia associated with thoracic and abdominal viscera, including the heart, lungs, and GI tract to the descending colon. Special sensory fibers from the nodose (inferior) ganglion of X carry information from taste buds in the posterior pharynx and send central branches to the rostral nucleus solitarius. Primary sensory axons from the inferior ganglion convey general sensation from the larynx, pharynx, and thoracic and abdominal viscera and terminate mainly in the caudal nucleus solitarius. Primary sensory axons from the jugular (superior) ganglion of X convey general sensation from the external auditory meatus and terminate in the spinal (descending) nucleus of V. A unilateral lesion of CN X, in addition to producing the motor symptoms, may result in ipsilateral laryngeal anesthesia and tachycardia or arrhythmias.

Accessory Nerve (CN XI)

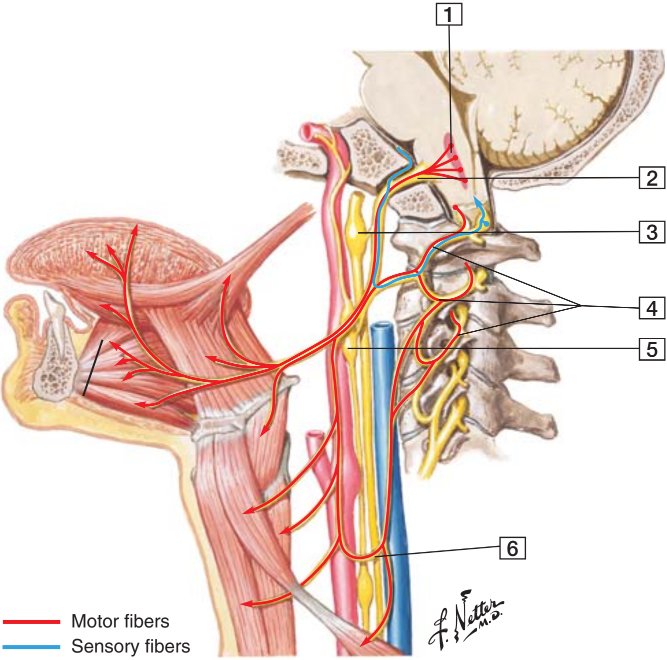


Accessory Nerve (CN XI)

1. Nucleus ambiguus
2. Vagus nerve (CN X)
3. Spinal root of CN XI
4. Foramen magnus
5. Jugular foramen
6. Superior (jugular) ganglion of CN X
7. Accessory nerve (CN XI)
8. Inferior (nodose) ganglion of CN X
9. First spinal nerve (C1)
10. External branch of CN XI
11. Sternocleidomastoid muscle
12. Trapezius muscle

Comment: The accessory nerve (CN XI) is a motor nerve with a cranial and a spinal portion. The cranial portion arises from lower motor neurons (LMNs) at the caudal end of nucleus ambiguus and travels with the pharyngeal and laryngeal branches of the vagus nerve and with nerves to the soft palate; some authors consider this to be part of CN X. The spinal portion arises from LMNs in the lateral part of the upper four to five cervical segments of the spinal cord; the axons emerge as rootlets laterally, ascend behind the denticulate ligaments, ascend through the foramen magnus, and join CN X to exit through the jugular foramen. These LMNs supply the sternocleidomastoid muscle and the upper two thirds of the trapezius muscle ipsilateral. Unilateral damage to CN XI results in weakness in head rotation (to the opposite side) and ipsilateral shoulder elevation. Bilateral lesions (sometimes seen in amyotrophic lateral sclerosis) result in inability to lift the head or shrug the shoulders.

Hypoglossal Nerve (CN XII)



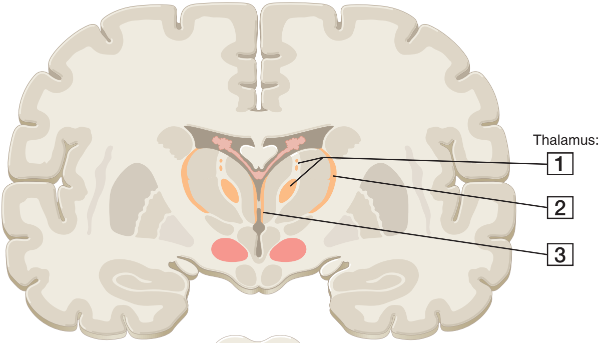
Hypoglossal Nerve (CN XII)

1. Hypoglossal nucleus
2. Hypoglossal nerve (CN XII)
3. Inferior (nodose) ganglion of CN X
4. Ventral rami of C1, C2, and C3 forming the cervical plexus
5. Superior cervical ganglion (sympathetic)
6. Ansa cervicalis (ansa hypoglossi)

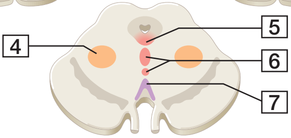
Comment: The hypoglossal nerve (CN XII) is a motor nerve. Lower motor neurons in the hypoglossal nucleus in the medulla exit from the ventral surface in the preolivary sulcus and innervate the extrinsic muscles of the tongue (hypoglossus, styloglossus, chondroglossus, and genioglossus) and the intrinsic muscles of the tongue (the superior and inferior longitudinal, transverse, and vertical lingual muscles). Damage to this nerve results in weakness of the ipsilateral tongue muscles, with muscle wasting. When it protrudes, the tongue deviates toward the weak side because of the unopposed action of the contralateral genioglossus muscles. During the initial phase of damage to the tongue from a lesion of CN XII, fasciculations may be seen on the ipsilateral tongue; these disappear with total denervation.

Reticular Formation and Nuclei

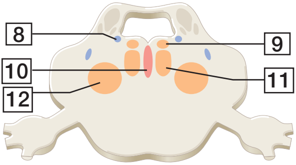
A. Thalamus and hypothalamus



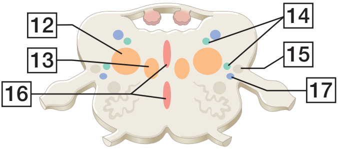
B. Midbrain



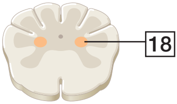
C. Pons



D. Medulla



E. Spinal cord–medullary junction



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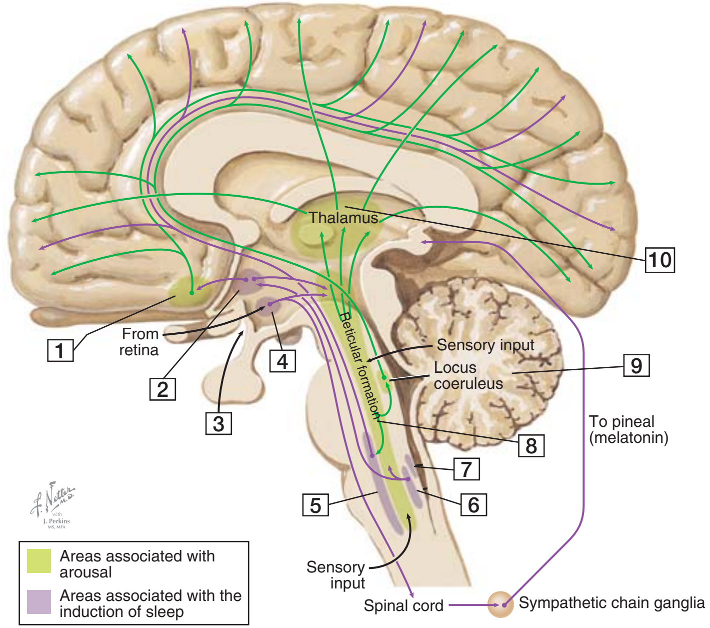
MS, MFA

Reticular Formation and Nuclei

1. Intralaminar nuclei (thalamus)
2. Reticular nucleus of the thalamus
3. Midline nuclei (thalamus)
4. Lateral reticular formation of the midbrain
5. Periaqueductal gray
6. Raphe nuclei (dorsal, central superior)
7. Ventral tegmental area
8. Locus caeruleus
9. Parapontine reticular formation (PPRF)—lateral gaze center
10. Raphe nuclei (pontis)
11. Pontine reticular formation (pontis caudalis, oralis)
12. Lateral reticular formation
13. Medullary reticular formation (gigantocellularis)
14. Respiratory nuclei
15. Rostral ventrolateral medulla (RVLM)
16. Raphe nuclei (obscurus, pallidus, magnus)
17. Group A1
18. Lamina 7—caudal reticular formation (RF)

Comment: The RF is the neuronal core of the brain stem, extending from the rostral spinal cord through the hypothalamus and septal region; RF neurons are large cells with axonal arborizations that terminate a distance from their cell bodies and dendritic trees (isodendritic). RF neurons are in a lateral zone (predominantly sensory functions), a medial zone (predominantly motor functions for tone and posture), a column of midline and lateral serotonergic neurons, and clusters of noradrenergic neurons. These two lateral groups of neurons exert modulatory influences on CXIS targets. The sensory zone of the RF is the *ascending reticular activation system* (ARAS); it receives multiple sensory inputs, activates the cortex through nonspecific thalamic nuclei, and participates in arousal, alertness, and consciousness. Consciousness requires intact ARAS and at least one functioning hemisphere. The medial motor portion of the RF consists of the pontine and medullary reticulospinal systems (upper motor neurons) and paramedian nuclei such as the PPRF (lateral gaze center).

Sleep-Wakefulness Control

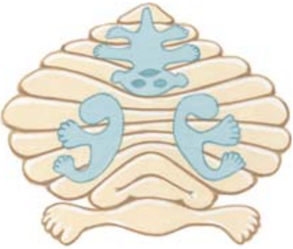


Sleep-Wakefulness Control

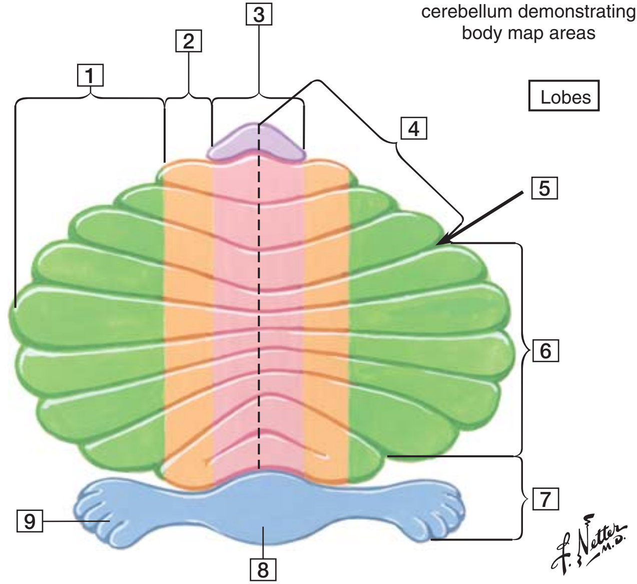
1. Nucleus basalis (cholinergic)
2. Preoptic hypothalamic area
3. Interleukins, other blood-borne substances
4. Suprachiasmatic nucleus
5. Raphe nuclei
6. Nucleus solitarius
7. Area postrema
8. Reticular formation
9. Locus coeruleus
10. Thalamus

Comment: Sleep is a normal physiological state involving a cyclic temporary loss of consciousness, readily reversed by sensory stimuli. Sleep is an active process initiated by brain activity in several regions, including the locus coeruleus, the raphe nuclei of the medulla and pons, nucleus solitarius, cholinergic neurons of the brain stem tegmentum, the lateral reticular formation (particularly the pons), several regions of the hypothalamus, and the reticular nucleus of the thalamus. Many of these regions actively inhibit the lateral sensory portion of the reticular formation that normally maintains wakefulness and consciousness. Circulating substances such as interleukin-1 can act on sites in the hypothalamus to induce sleepiness (a component of illness behavior). Slow wave sleep that does not involve rapid eye movement (REM) is initiated by hypothalamic neurons and is accompanied by decreased activity in the locus coeruleus and the cholinergic tegmental neurons. During REM sleep, activity in the noradrenergic locus coeruleus and the serotonergic raphe neurons greatly diminishes, preventing the cerebral cortex from attending to external stimuli. Dreams probably occur because the cortex is attending to internal stimuli provided by stored memories.

Cerebellar Organization: Lobes and Regions



“Unfolded” schematic of cerebellum demonstrating body map areas



“Unfolded” schematic of cerebellum demonstrating regions and lobes

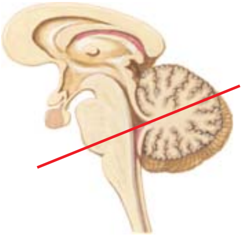
Cerebellar Organization: Lobes and Regions

1. Lateral hemisphere
2. Paravermis
3. Vermis
4. Anterior lobe
5. Primary fissure
6. Posterior lobe
7. Flocculonodular lobe
8. Nodule
9. Flocculus

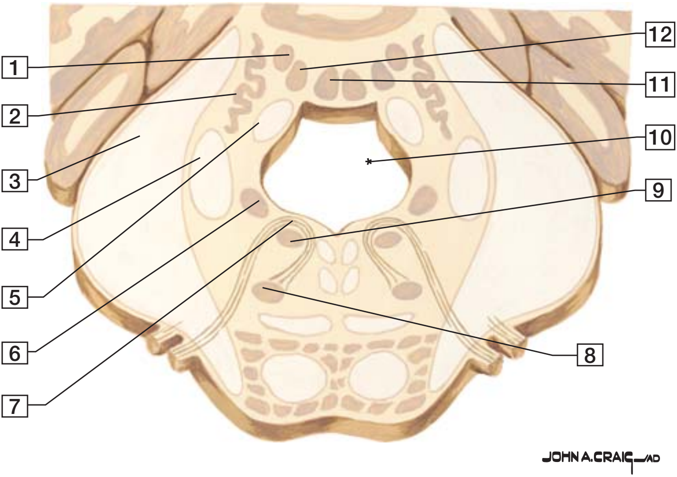
Comment: The cerebellum is subdivided by lobes (anterior, posterior, flocculonodular) and longitudinal organization (vermis, paravermis, lateral hemispheres). Damage to the flocculonodular lobe produces truncal ataxia and a broad-based uncoordinated gait. Damage confined to the anterior lobe results in a staggering gait with stiff-legged movement, brought about by disinhibition of anterior lobe connections with the lateral vestibulospinal system. Posterior lobe damage (affecting lateral hemispheres) produces additional problems beyond truncal ataxia, including limb ataxia, dysmetria, dysdiadochokinesia (inability to perform rapid alternating movements), intention tremor, and hypotonia. The longitudinal organization reflects connectivity of the hemispheres with the deep cerebellar nuclei and their influence over upper motor neuronal groups. The vermis and flocculonodular lobe connect with the fastigial nucleus and lateral vestibular nucleus, which regulate vestibulospinal and reticulospinal tract activity (ipsilateral). The paravermis connects with globose and emboliform nuclei, which regulate mainly rubrospinal tract activity. The lateral cerebellar hemispheres connect with the dentate nucleus, which regulates mainly corticospinal tract activity via the ventral lateral nucleus of the thalamus. These two connections from the deep nuclei to their upper motor neurons (UMNs) are crossed, and UMN projections are crossed. Cerebellar influences over motor activity are ipsilateral, as are the symptoms that occur with damage.

See book 11.34

Cerebellar Anatomy: Deep Nuclei and Cerebellar Peduncles



Level of section



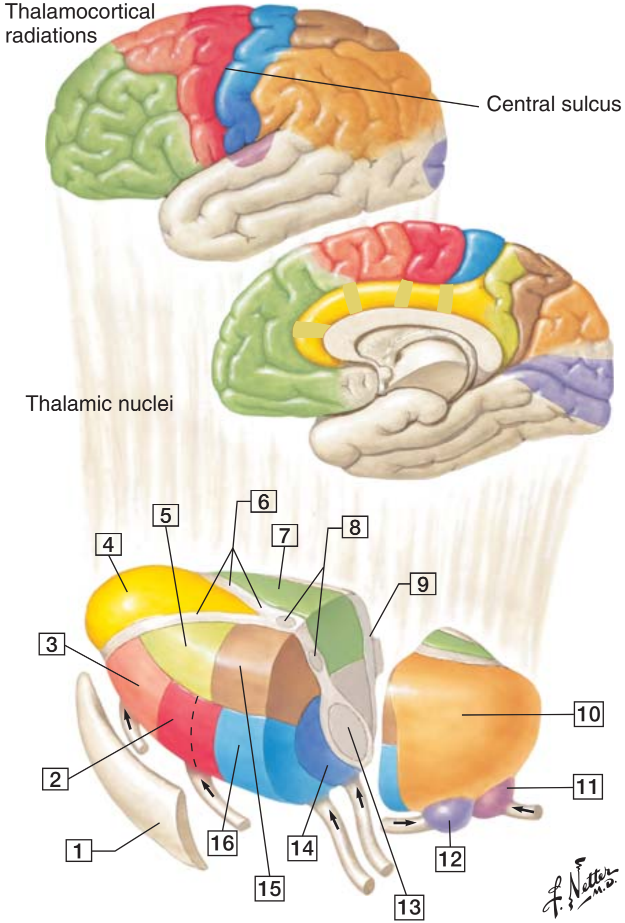
Cerebellar Anatomy: Deep Nuclei and Cerebellar Peduncles

1. Emboliform nucleus
2. Dentate nucleus
3. Middle cerebellar peduncle (MCP)
4. Inferior cerebellar peduncle (ICP)
5. Superior cerebellar peduncle (SCP)
6. Lateral vestibular nucleus
7. Genu of the facial nerve (CN VII)
8. Nucleus of CN VII (facial)
9. Nucleus of CN VI (abducens)
10. Fourth ventricle
11. Fastigial nucleus
12. Globose nucleus

Comment: The ICP conveys extensive afferents to the cerebellum (cortex and deep nuclei) from the spinocerebellar system, reticular formation, vestibular system, and trigeminal system; it also conveys efferents from the fastigial nucleus and the flocculonodular lobe to upper motor neusons (UMNs) of the vestibulospinal and reticulospinal systems. The MCP mainly conveys afferents to the cerebellum from the corticopontocerebellar system. The SCP conveys selective afferents to the cerebellum from the ventral spinocerebellar tract, some trigeminal input, tectocerebellar input, and coeruleocerebellar noradrenergic projections; it also conveys extensive efferents from the globose, emboliform, and dentate nuclei to the red nucleus and ventral lateral thalamus to regulate the rubrospinal and corticospinal UMN systems.

The vascular supply to the cerebellum comes mainly from the superior, anterior inferior, and posterior inferior cerebellar arteries. The cerebellum is prone to intracerebral bleeds and hematomas. Damage from specific focal lesions is ipsilateral; space-occupying masses may induce herniation and more general symptoms of increased intracranial pressure.

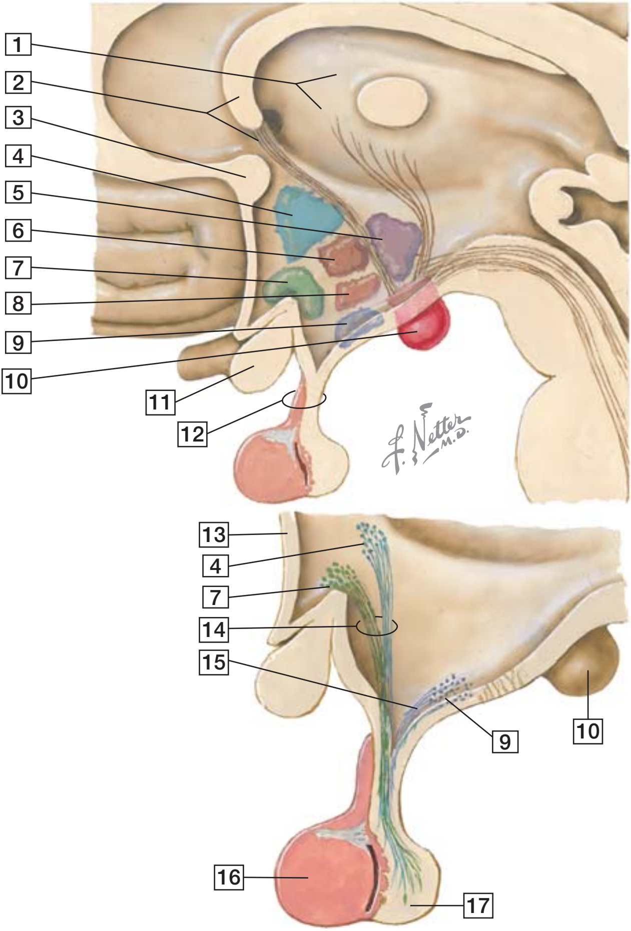
Thalamic Anatomy and Interconnections with the Cerebral Cortex



Thalamic Anatomy and Interconnections with the Cerebral Cortex

1. Reticular nucleus
2. Ventral lateral (VL)
3. Ventral anterior (VA)
4. Anterior nuclei
5. Lateral dorsal (LD)
6. Internal medullary lamina
7. Medial dorsal (MD)
8. Intralaminar nuclei
9. Midline (median) nuclei
10. Pulvinar
11. Medial geniculate nucleus (body)
12. Lateral geniculate nucleus (body)
13. Centromedian (CM)
14. Ventral posteromedial (VPM)
15. Lateral posterior (LP)
16. Ventral posterolateral (VPL)

Comment: The thalamus conveys extensive sensory, motor, and autonomic information from the spinal cord and brain stem to the cerebral cortex. All sensory systems are processed through the thalamus except olfaction. Most thalamic nuclei are reciprocally connected with the cerebral cortex. These nuclei are categorized as sensory projection nuclei (VPL—somatosensory; VPM—trigeminal; LGN—visual; MGN—auditory; pulvinar—sensory); motor-related projection nuclei (VL and VI—cerebellum; VA and VL—basal ganglia); autonomic- and limbic-related projection nuclei (anterior and LD—cingulate cortex; MD—frontal and cingulate cortex); and association area–related projection nuclei (pulvinar and LP—parietal cortex). Nonspecific thalamic nuclei (CM, parafascicular, medial VA) send diffuse connections to widespread cortical areas and to other thalamic nuclei. The reticular nucleus helps to regulate the excitability of other thalamic projection nuclei.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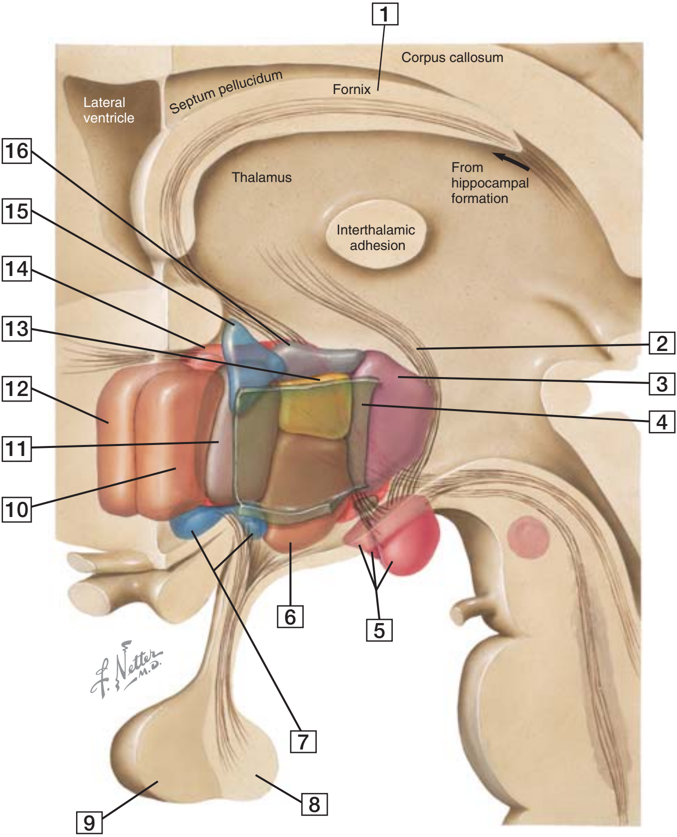
Hypothalamus and Pituitary Gland

1. Thalamus
2. Fornix
3. Anterior commissure
4. Paraventricular nucleus
5. Posterior area
6. Dorsomedial nucleus
7. Supraoptic nucleus
8. Ventromedial nucleus
9. Arcuate nucleus
10. Mammillary nuclei (body)
11. Optic chiasm
12. Infundibulum (pituitary stalk)
13. Lamina terminalis
14. Supraopticohypophyseal tract
15. Tuberohypophyseal tract
16. Adenohypophysis (anterior lobe of pituitary)
17. Neurohypophysis (posterior lobe of pituitary)

Comment: The hypothalamus is the major region of the CNS involved in neuroendocrine regulation and control of visceral functions such as temperature, appetite, thirst and water balance, reproduction and sexual behavior, parturition and lactation, respiratory and cardiovascular regulation, gastrointestinal regulation, stress responses, reparative states, and immune regulation. The neuroendocrine portion of the hypothalamus consists of (1) the magnocellular paraventricular (PVN) and supraoptic (SON) nuclei, which send axons directly to the posterior pituitary and release oxytocin and vasopressin into the general circulation; (2) releasing-factor and inhibitory-factor neurons, which project axons to the hypophysial-portal vasculature in the contact zone of the median eminence; and (3) tuberoinfundibular system and ascending systems (monoamines and other chemically specific neurons) that modulate release of releasing- and inhibitory-factor neurons into the hypophysial-portal vasculature.

Hypothalamic Nuclei

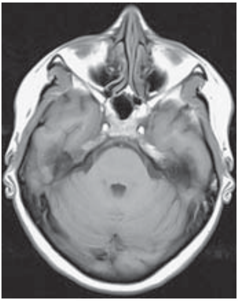


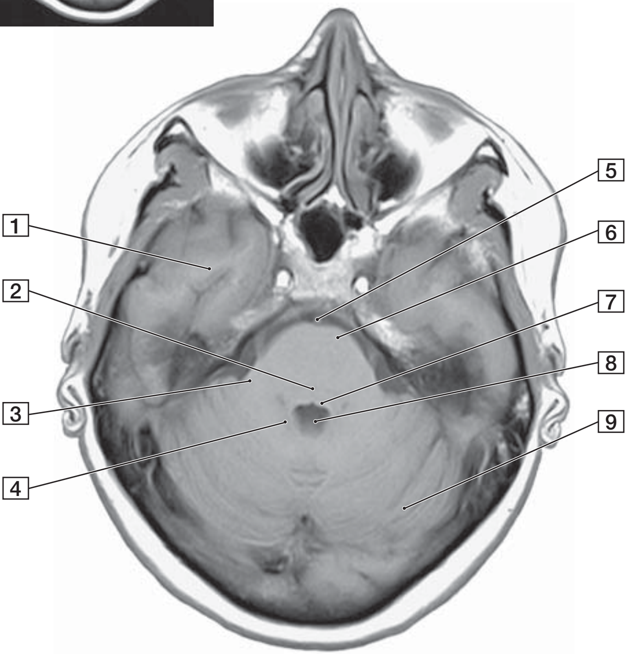
Hypothalamic Nuclei

1. Fornix
2. Mammillothalamic tract
3. Posterior hypothalamic area
4. Periventricular nucleus
5. Mammillary nuclei (complex)
6. Ventromedial nucleus
7. Supraoptic nucleus
8. Posterior lobe of the pituitary gland
9. Anterior lobe of the pituitary gland
10. Medial preoptic nucleus
11. Anterior hypothalamic area
12. Lateral preoptic nucleus
13. Dorsomedial nucleus
14. Lateral hypothalamic area
15. Paraventricular nucleus
16. Dorsal hypothalamic area

Comment: Hypothalamic nuclei and areas are associated with specific visceral or neuroendocrine functions. The paraventricular (PVN) and supraoptic magnocellular neurons are the source of oxytocin and vasopressin, released into general circulation in the posterior pituitary gland. Some parvocellular neurons of the PVN send descending axons to regulate autonomic preganglionic outflow from both sympathetic and parasympathetic nervous systems. Anterior and posterior areas coordinate parasympathetic and sympathetic outflow, respectively. The dorsomedial and ventromedial nuclei, as well as the lateral hypothalamic area, regulate appetite, drinking, and reproductive behavior. The preoptic area regulates cyclic neuroendocrine behavior and thermoregulation. The suprachiasmatic nucleus receives visual inputs from the optic tract and regulates circadian rhythms and diurnal cycles. Several hypothalamic regions are involved in the regulation of sleep. The concept of "centers" and "areas" of hypothalamic nuclei is complicated by the abundant presence of passing axonal tracts affected in early studies. Interleukins, such as IL-1 beta, IL-6, and TNF-alpha, can act on hypothalamic regions and induce changes in neuroendocrine outflow, sympathetic and parasympathetic control of stress hormones, thermoregulation, affective behavior, and other functions.

Axial Sections through the Forebrain: Midpons





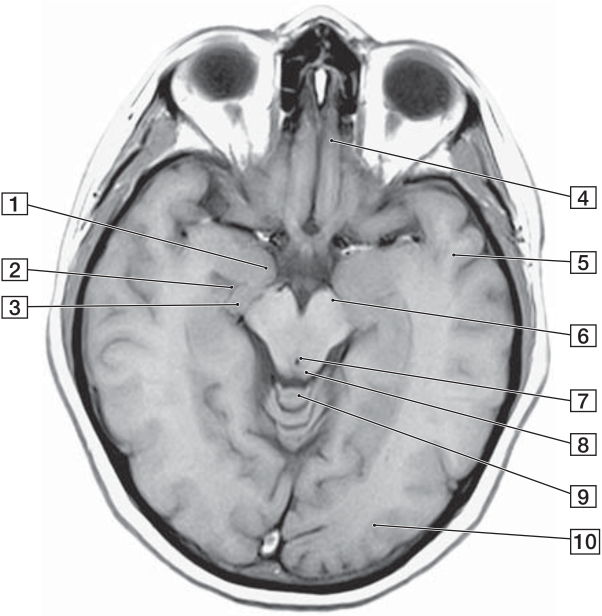
Axial Sections through the Forebrain: Midpons

1. Temporal lobe
2. Pontine tegmentum
3. Middle cerebellar peduncle
4. Superior cerebellar peduncle
5. Basilar artery
6. Basis pontis with corticospinal tract
7. Medial longitudinal fasciculus
8. Fourth ventricle
9. Lateral cerebellar hemisphere

Comment: An axial section through the posterior portion of the forebrain reveals the fundamental organization of the brain stem. The corticospinal system is located anteriorly in the brain stem, including the cerebral peduncles in the midbrain, the basis pontis in the pons, and the pyramids in the medulla. At this level, in the pons, the corticospinal system is broken up into individual fascicles; intermixed among these fascicles are neurons of the pontine nuclei, which channel corticopontocerebellar feedback from the cerebral cortex to the cerebellum. In a midposition is the brain stem tegmentum, containing the cranial nerves and their nuclei, the reticular formation, the major ascending sensory channels, and the autonomic nuclei and their channels. Located most posteriorly is the cerebellum, particularly its hemispheres with their folia. The ventricular system (fourth ventricle, aqueduct) is found in the dorsal (posterior) tegmentum and at the base of the cerebellum. The deep cerebellar nuclei are found just above the roof of the fourth ventricle. A mass lesion in the posterior fossa may induce cerebellar symptoms; brain stem–related problems (nausea, vomiting); headache; obtunded consciousness; and a threat of herniation of the brain stem through the foramen magnum, a potentially fatal event.

See book 13.1B

Axial Sections through the Forebrain: Midbrain

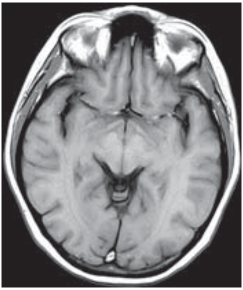


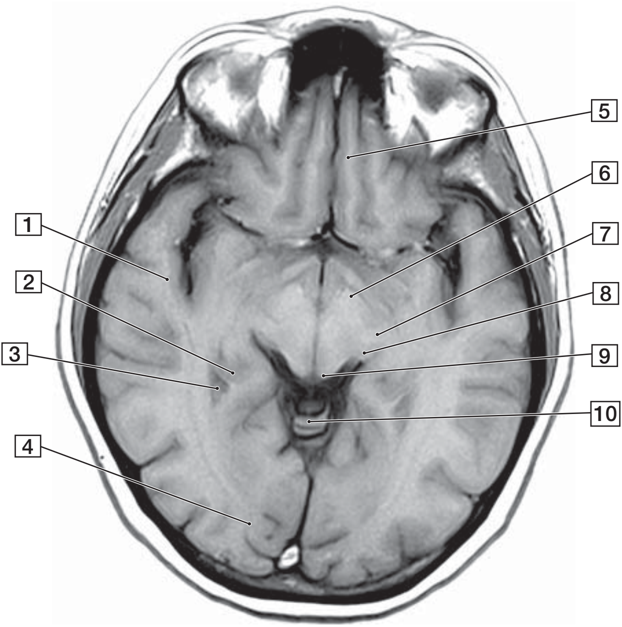
Axial Sections through the Forebrain: Midbrain

1. Amygdala
2. Inferior horn of the lateral ventricle
3. Hippocampal formation
4. Orbitofrontal cortex
5. Temporal lobe
6. Cerebral peduncle
7. Aqueduct
8. Superior colliculus
9. Cerebellar vermis
10. Occipital lobe

Comment: The temporal lobe includes the amygdaloid nuclei; the hippocampal formation and associated cortex (parahippocampal, entorhinal); the transverse gyrus of Heschl and associated auditory cortex; some language-associated cortical regions (Wernicke's area for receptive language function in the dominant hemisphere); Meyer's loop of the geniculocalcarine axons; the inferior horn of the lateral ventricle; and extensive cortical areas, particularly association areas (superior, middle, and inferior temporal gyri). The temporal lobe is susceptible to damage by trauma, infarcts, tumors, abscesses, and other pathological conditions such as seizures. Such damage may result in auditory hallucinations, delirium, and psychotic behavior. If Meyer's loop is damaged, an upper contralateral quadrantanopia may occur. If Wernicke's area is damaged in the dominant hemisphere, a receptive aphasia may occur, with inability to understand verbal information. Some specific lacunar infarcts may induce specific agnosias, such as the inability to recognize faces (prosopagnosia) or to distinguish animate from inanimate objects.

Axial Sections through the Forebrain: Rostral Midbrain and Hypothalamus



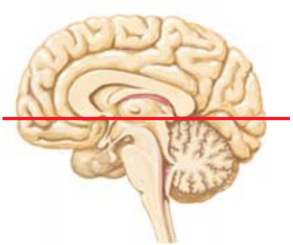


Axial Sections through the Forebrain: Rostral Midbrain and Hypothalamus

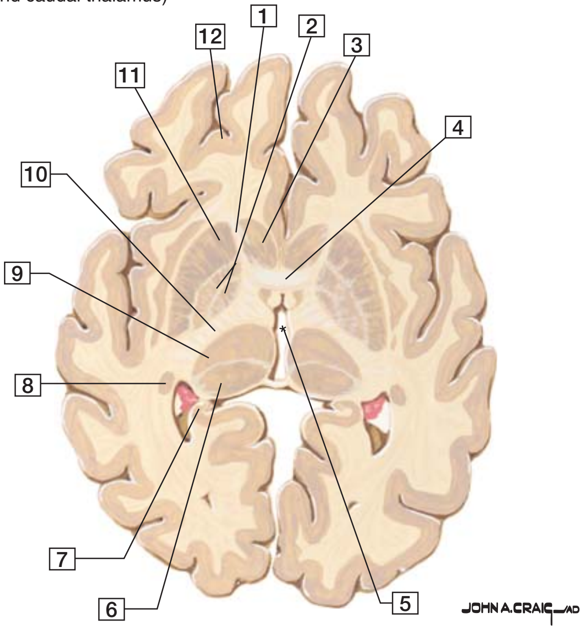
1. Temporal lobe
2. Hippocampal formation
3. Temporal horn of the lateral ventricle
4. Occipital lobe
5. Orbitofrontal cortex
6. Cerebral peduncle
7. Lateral geniculate nucleus
8. Medial geniculate nucleus
9. Superior colliculus
10. Cerebellar vermis

Comment: The transition between midbrain and caudal diencephalon reveals several key structures bridging brain stem and forebrain. The substantia nigra contains dopamine neurons that project to the caudate nucleus and putamen (via the nigrostriatal tract) and plays a key role in regulating basal ganglia function; degeneration of these dopamine neurons results in Parkinson's disease, characterized by resting tremor, muscular rigidity through all ranges of motion, bradykinesia, and postural instability. Geniculate nuclei are the most caudal of specific thalamic nuclei; the lateral geniculate nucleus receives optic tract information and provides the geniculocalcarine pathway for interpretation of the visual world, and the medial geniculate nucleus receives auditory input from the inferior colliculus and other brain stem auditory nuclei and provides the auditory radiations for sound interpretation. The superior colliculus is a visual structure that receives visual input from the optic tract and provides a supplemental visual pathway to visual association cortices, as well as a reflex pathway for visually related reflexes. The red nucleus is an important interconnecting structure for the basal ganglia, is the origin of the rubrospinal tract for regulating some flexor movements of the limbs, and is a secondary component of cortical control over brain stem upper motor neurons.

Axial Sections through the Forebrain: Anterior Commissure and Caudal Thalamus



Level of section
(anterior commissure
and caudal thalamus)

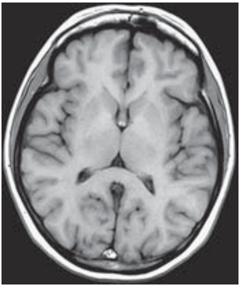


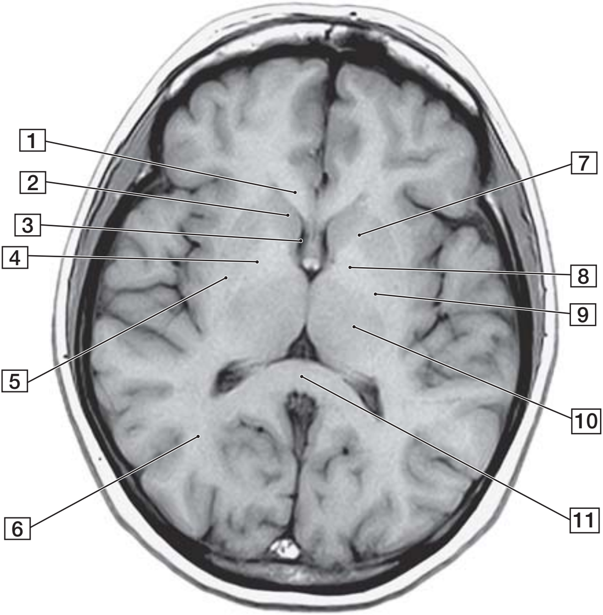
Axial Sections through the Forebrain: Anterior Commissure and Caudal Thalamus

1. Anterior limb of the internal capsule
2. Globus pallidus (internal and external segments)
3. Head of the caudate nucleus
4. Anterior commissure
5. Third ventricle
6. Pulvinar
7. Hippocampal formation
8. Tail of the caudate nucleus
9. Thalamus
10. Posterior limb of the internal capsule
11. Putamen
12. Orbitofrontal cortex

Comment: The basal ganglia assist the cerebral cortex in planning and generating desired programs of activity and in suppressing nondesired programs of activity; this role is most conspicuous for motor regulation. Basal ganglia lesions produce movement disorders that are often involuntary in nature and may be accompanied by cognitive and affective symptoms (e.g., Huntington's disease). The principal interconnections of the basal ganglia are from the thalamus and cerebral cortex to the striatum (caudate nucleus and putamen), then to the globus pallidus, and from the globus pallidus back to the thalamus and cortex, completing the loop. The subthalamus (a diencephalic structure) interconnects with the globus pallidus, and the substantia nigra (a midbrain structure) interconnects with the striatum. Disruption of these basal ganglia loops can produce excessive movement (e.g., choreiform [brisk dancelike] movements, athetosis [writhing movements], dystonia [a fixed abnormal posture], or ballismus [wild flinging movements] seen with subthalamic nucleus lesions).

Axial Sections through the Forebrain: Head of the Caudate Nucleus and Midthalamus



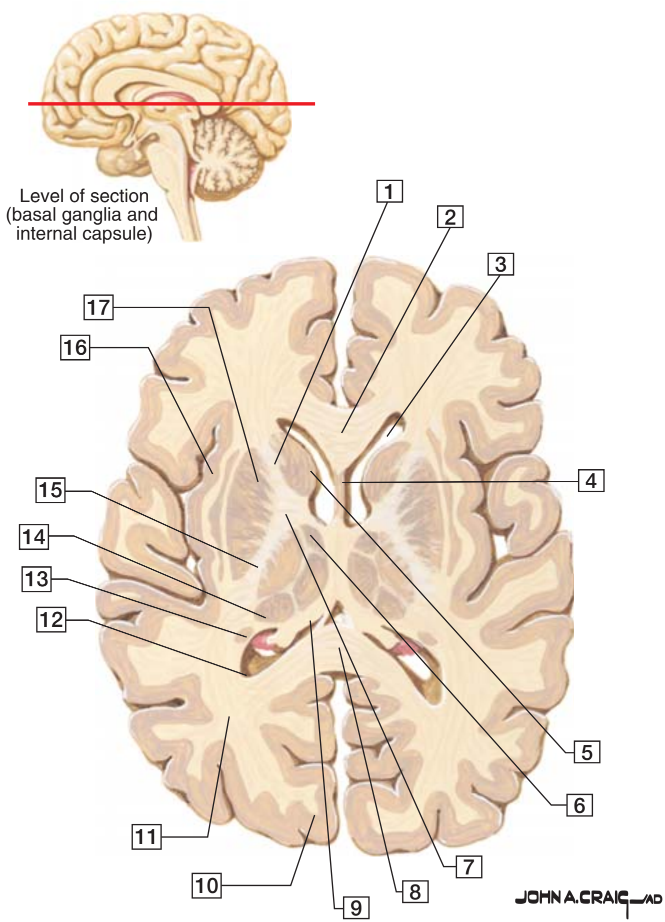


Axial Sections through the Forebrain: Head of the Caudate Nucleus and Midthalamus

1. Genu of the corpus callosum
2. Head of the caudate nucleus
3. Anterior horn of the lateral ventricle
4. Globus pallidus
5. Putamen
6. Optic radiations
7. Anterior limb of the internal capsule
8. Genu of the internal capsule
9. Posterior limb of the internal capsule
10. Thalamus
11. Splenium of the corpus callosum

Comment: The internal capsule reciprocally interconnects cortex and more caudal structures. The anterior limb carries cortical projections to the striatum and pontine nuclei (corticopontocerebellar system) and has reciprocal connections with important thalamic structures such as the medial dorsal and anterior nuclei. The genu carries corticobulbar axons destined for brain stem nuclei regulation, particularly motor cranial nerve nuclei. The posterior limb conveys major descending corticospinal, corticorubral, and corticoreticular fibers; major ascending sensory connections for the somatosensory and trigeminal system; and, posteriorly, visual and auditory radiations. The internal capsule is susceptible to middle cerebral artery lesions, particularly narrow lenticulostriate arteries. A vascular lesion of the posterior limb of the internal capsule results (long term, after resolution of the flaccid stage of shock) in contralateral spastic hemiplegia with hypertonus, hyperreflexia, flexed posture of the upper limb and extended and internally rotated posture of the lower limb, and pathological reflexes (e.g., plantar extensor response, possibly clonus). Such a lesion also produces loss of sensation on the contralateral body. If the genu of the internal capsule is involved in the lesion, a contralateral drooping lower face (central facial palsy) is seem due to removal of control of the corticobulbar system from regulation of the contralateral facial nerve motor nucleus for muscles of facial expression for the lower face.

Axial Sections through the Forebrain: Basal Ganglia and Internal Capsule

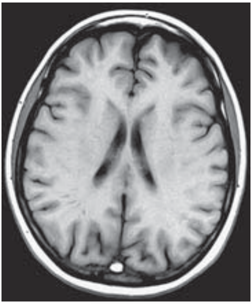


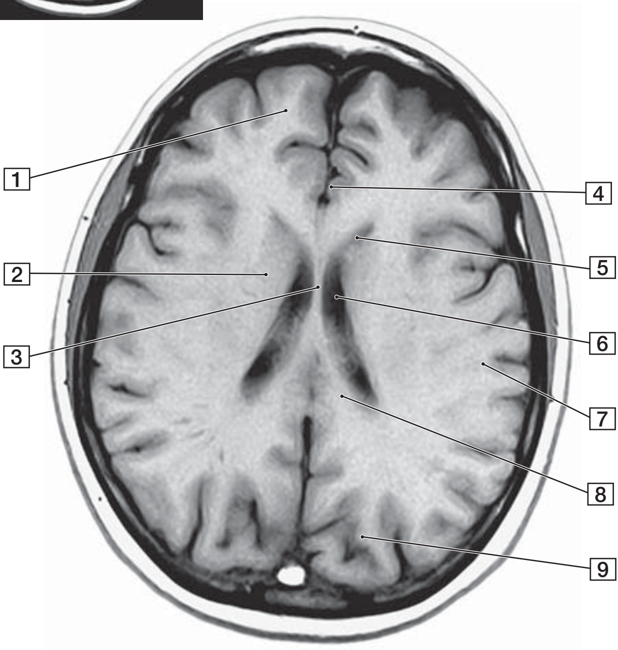
Axial Sections through the Forebrain: Basal Ganglia and Internal Capsule

1. Anterior limb of the internal capsule
2. Rostrum of the corpus callosum
3. Frontal pole of the lateral ventricle
4. Septum pellucidum
5. Head of the caudate nucleus
6. Anterior thalamic nucleus
7. Genu of the internal capsule
8. Splenium of the corpus callosum
9. Body of the fornix
10. Occipital lobe
11. Optic radiations
12. Temporal pole of the lateral ventricle
13. Tail of the caudate nucleus
14. Pulvinar
15. Posterior limb of the internal capsule
16. Insular cortex
17. Putamen

Comment: Huntington's disease is an autosomal dominant disorder caused by a trinucleotide repeat (CAG) on the short arm of chromosome 4. This disorder results in a progressive, untreatable disease that includes a movement disorder (choreiform movements—brisk, jerky, forcible, arrhythmic movements); a progressive cognitive impairment; and affective problems (depression, psychotic behavior). Huntington's disease may progress from a state of minor impairment (clumsiness, irritability, depression) to major impairment, incapacitation, and an early death. The anatomical hallmark of this disease is the marked degeneration of the caudate nucleus (conspicuous on MR imaging) and also the putamen. The medium spiny neurons of the striatum that project to the pallidum degenerate because of excitotoxic damage (activation of N-methyl D-aspartate [NMDA] receptors that induce neuronal apoptosis by an influx of excessive Ca^{++}). The intrinsic cholinergic neurons of the striatum also degenerate.

Axial Section through the Forebrain: Dorsal Caudate Nucleus, Splenium and Genu of the Internal Capsule



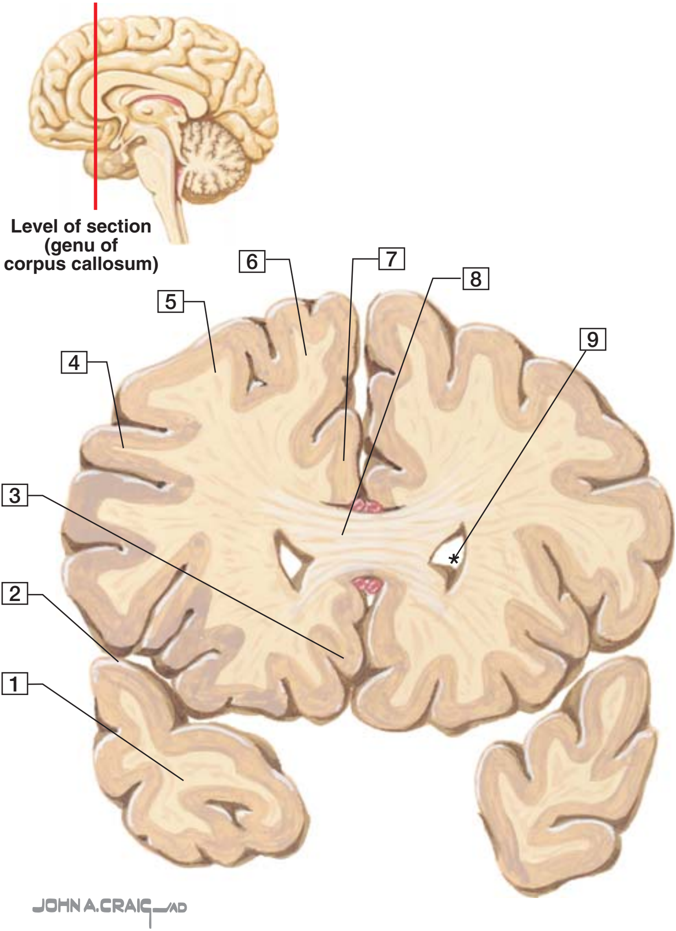


Axial Section through the Forebrain: Dorsal Caudate Nucleus, Splenium and Genu of the Internal Capsule

1. Frontal lobe
2. Body of the caudate nucleus
3. Septum pellucidum
4. Cingulate cortex
5. Frontal pole of the lateral ventricle
6. Body of the lateral ventricle
7. Parietal lobe
8. Splenium of the corpus callosum
9. Occipital lobe

Comment: The forebrain contains four structures and their associated pathways that reveal a C-shaped configuration reminiscent of their neuronal development from the lamina terminalis (rostral end of the neural tube) with migration from a frontal position to a more posterior and then caudal and anterior position into the temporal lobe. These systems are (1) the lateral ventricles that sweep in a C shape from the frontal pole to the body to the occipital horn, to the trigone area to the temporal pole. This section reveals the body; more posterior sections cut through the lateral ventricles in opposite poles; (2) the caudate nucleus, with its major bulk in the head, bulging into the frontal pole of the lateral ventricle, its more slender body revealed in this section, and a small tail that appears in the temporal lobe near the temporal pole of the lateral ventricle; (3) the hippocampal formation with its fornix, including the fimbria, the crura, the body, and the columns that sweep downward in the frontal lobe to terminate in the hypothalamus and septum; and (4) the amygdaloid complex and its associated pathway, the stria terminalis, that also sweeps forward in a C shape to terminate in the hypothalamus.

Coronal Sections through the Forebrain: Genu of the Corpus Callosum

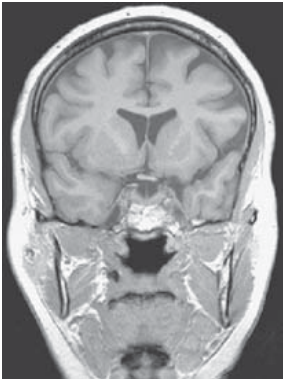


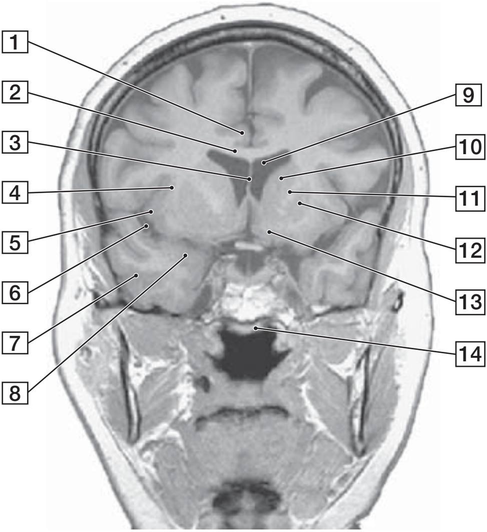
Coronal Sections through the Forebrain: Genu of the Corpus Callosum

1. Temporal pole
2. Lateral fissure
3. Subcallosal gyrus
4. Inferior frontal gyrus
5. Middle frontal gyrus
6. Superior frontal gyrus
7. Cingulate gyrus
8. Genu of the corpus callosum
9. Frontal pole of the lateral ventricle (asterisk)

Comment: The prefrontal cortex subserves an important role in anticipatory behavior and in the process of socialization. This cortex allows us to contemplate long-term consequences of actions (goal orientation), to learn from the past, to anticipate the future and work diligently, even in difficult circumstances, for the hope of a better future. The process of conscience and doing what is deemed socially right also depends on some frontal cortical function. A specialized region of the inferior frontal gyrus in the dominant hemisphere (usually the left), just above the rostral portion of the lateral fissure, is a region for expressive language function, including both spoken word and written word. If Broca's area is damaged by a vascular infarct (middle cerebral artery) on the dominant side, the patient will experience expressive aphasia and have great difficulty uttering words and initiating language function.

Coronal Sections through the Forebrain: Head of the Caudate Nucleus and Nucleus Accumbens



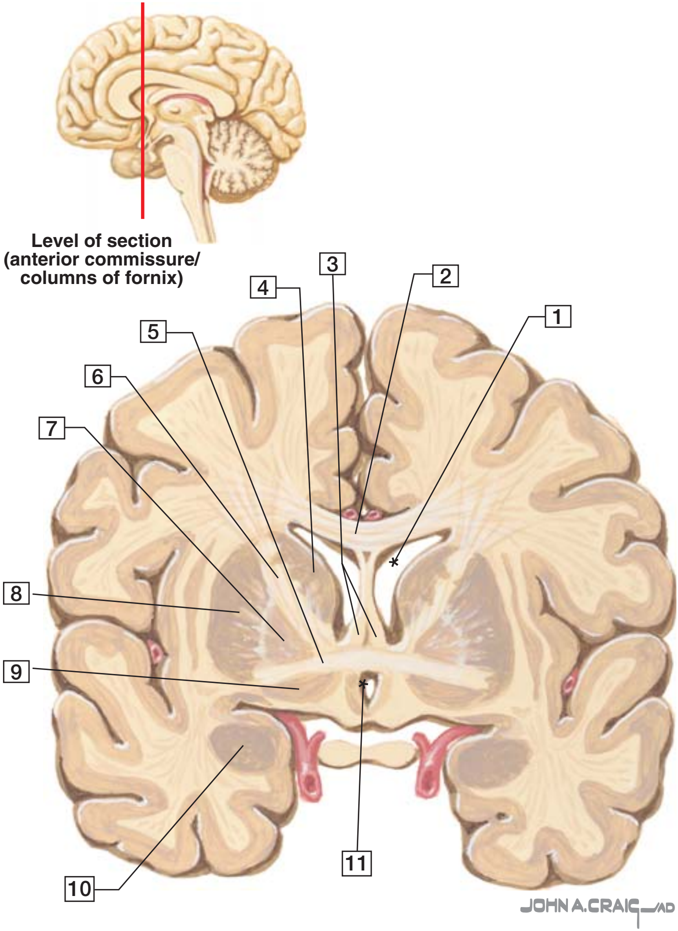


Coronal Sections through the Forebrain: Head of the Caudate Nucleus and Nucleus Accumbens

1. Cingulate gyrus
2. Body of the corpus callosum
3. Septum pellucidum
4. Claustrum
5. Insular cortex
6. Lateral fissure
7. Temporal lobe
8. Amygdala
9. Frontal pole of the lateral ventricle
10. Head of the caudate nucleus
11. Anterior limb of the internal capsule
12. Putamen
13. Nucleus accumbens
14. Optic chiasm

Comment: The nucleus accumbens is found at the anterior end of the striatum in the ventral part of the forebrain anterior to the anterior limb of the internal capsule. It receives extensive inputs from limbic structures such as the amygdala, the hippocampal formation, and the bed nucleus of the stria terminalis. Major dopaminergic inputs from the ventral tegmental area of the midbrain travel via the mesolimbic dopaminergic pathway to supply the nucleus accumbens with a dense bed of terminals. The nucleus accumbens is a key structure for motivational states and addictive behaviors, driven by dopaminergic neurotransmission. The nucleus accumbens also is a principal region for brain circuitry associated with reward, such as joy, pleasure, and gratification. This nucleus has looped circuitry through the thalamus and cortex that contributes to motor expression of emotional responses and their accompanying gestures and behaviors.

Coronal Sections through the Forebrain: Anterior Commissure and Columns of the Fornix



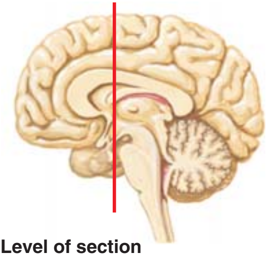
Coronal Sections through the Forebrain: Anterior Commissure and Columns of the Fornix

1. Frontal pole of the lateral ventricle
2. Body of the corpus callosum
3. Columns of the fornix
4. Head of the caudate nucleus
5. Anterior commissure
6. Anterior limb of the internal capsule
7. Globus pallidus
8. Putamen
9. Nucleus basalis
10. Amygdala
11. Third ventricle (asterisk)

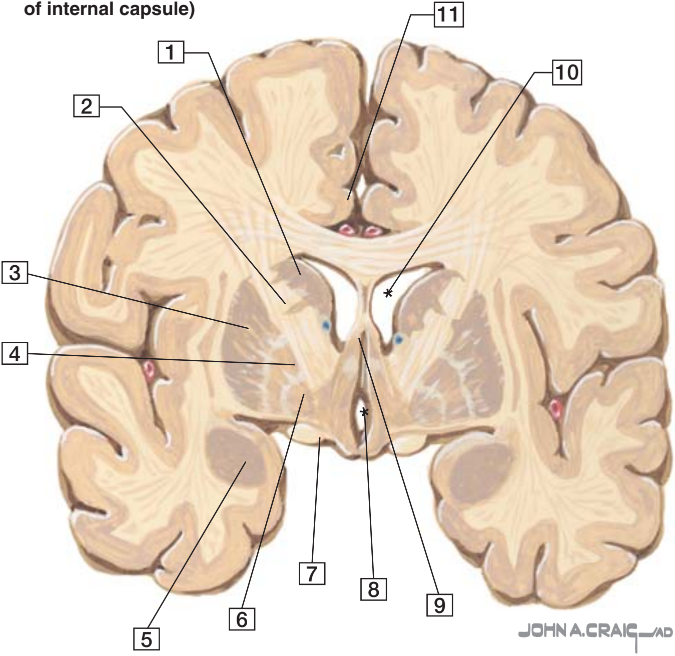
Comment: The head of the caudate nucleus is found medial to the anterior limb of the internal capsule, and the putamen and globus pallidus are found lateral to the anterior limb. The caudate nucleus and putamen are similar in neuronal composition and, in most mammals, are represented by a single nucleus, the caudoputamen; in humans, this complex has been subdivided by the intervention of the robust internal capsule. The globus pallidus, found ventrally and medially in the compartment lateral to the anterior limb, is the major component of the basal ganglia that carries outflow to the thalamus. It is bounded ventrally by the anterior commissure. Ventral to the anterior commissure is the substantia innominata; a major nuclear component is the nucleus basalis, which contains a cholinergic group of neurons that is important for memory function. This region deteriorates in some patients with Alzheimer's disease and may contribute to cognitive decline and memory dysfunction. Further ventral to the anterior commissure and nucleus basalis are the medial temporal lobe structures, such as the amygdala in the anterior region; this complex of nuclei is important in providing an emotional context for sensory stimuli and is particularly important in generating fear responses. Bilateral lesions in humans results in an inability to recognize fearful facial expressions in others.

See book 13.13A

Coronal Sections through the Forebrain: Amygdala, Anterior Limb of the Internal Capsule



Level of section
(amygdala, anterior limb
of internal capsule)

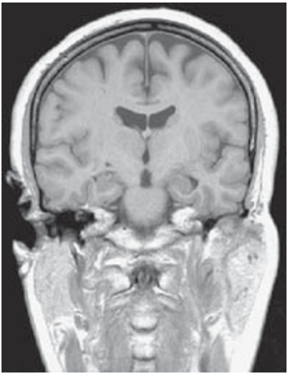


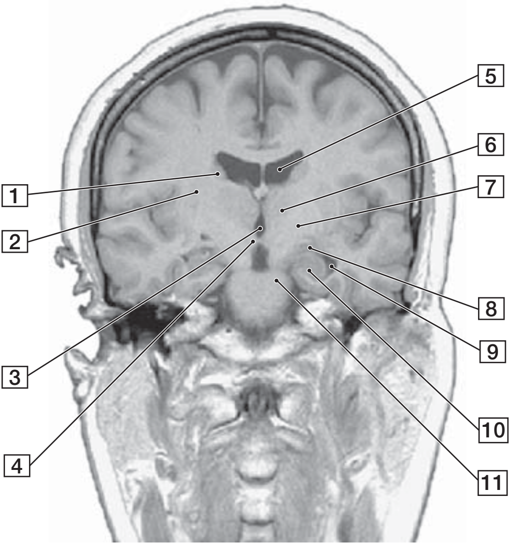
Coronal Sections through the Forebrain: Amygdala, Anterior Limb of the Internal Capsule

1. Body of the caudate nucleus
2. Anterior limb of the internal capsule
3. Putamen
4. Globus pallidus, external segment
5. Amygdala
6. Globus pallidus, internal segment
7. Optic tract
8. Third ventricle (asterisk)
9. Columns of the fornix
10. Body of the lateral ventricle (asterisk)
11. Cingulate cortex

Comment: The corpus callosum is the principal commissural (interhemispheric) pathway in the brain, connecting cortical regions on one side with their counterpart on the other side. The anterior commissure subserves this role for interconnections between portions of the temporal lobes. On some occasions, the corpus callosum (and anterior commissure) is surgically sectioned to prevent the spread of seizure activity from one side to the other; this is called a "split brain" preparation. When isolated by sectioning of the commissural pathways, each hemisphere is independent and is not aware of specific activity undertaken by the other side. Thus, the left hemisphere can identify visual stimuli presented in the right visual world, but not visual stimuli presented in the left visual world (processed by the right hemisphere). Similarly, the left hemisphere does not know where the left hand and arm are located if they are kept from the view of the left hemisphere; the left hand may act independently of the conscious intent of the left hemisphere. Some emotion content from the hemispheres may transfer to the other side through brain stem regions, providing a limbic context that may be perceived to some extent by both hemispheres.

Coronal Sections through the Forebrain: Mammillary Bodies



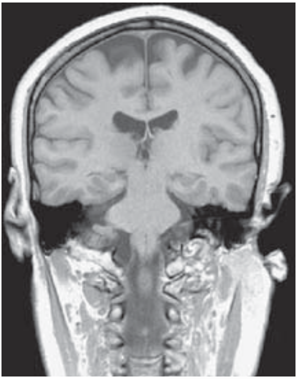


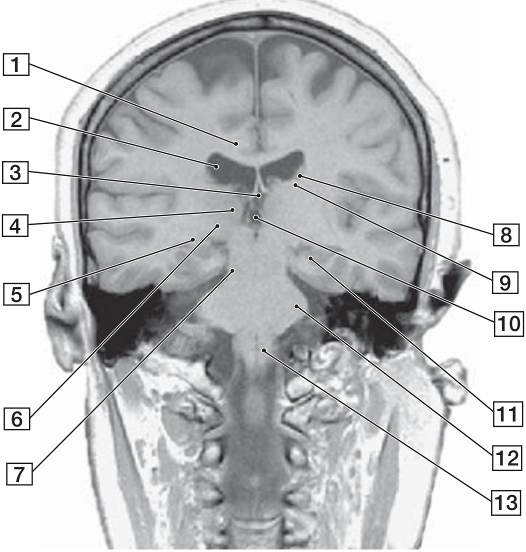
Coronal Sections through the Forebrain: Mammillary Bodies

1. Body of the caudate nucleus
2. Putamen
3. Third ventricle
4. Hypothalamus
5. Body of the lateral ventricle
6. Rostral thalamus
7. Posterior limb of the internal capsule
8. Amygdala
9. Inferior horn of the lateral ventricle
10. Hippocampal formation
11. Corticospinal tract in the basis pontis

Comment: Commissural pathways: Cortical interconnections between two hemispheres travel through corpus callosum and anterior commissure (portions of the temporal lobe). Association pathways: Cortical interconnections between close or distant regions of the same hemisphere travel through short and long association pathways, respectively. Projection pathways: Extensive cortical outflow travels from the cerebral cortex to subcortical structures through the internal capsule. Motor-related connections include the corticospinal tract, corticorubrospinal system, the corticoreticulospinal system (posterior limb), and corticobulbar system (genu). Cerebellar-related connections include the corticopontocerebellar system, derived from widespread cortical regions (anterior and posterior limbs). Basal ganglia–related connections include corticostriate, corticonigral, and corticosubthalamic connections (anterior and posterior limbs). Corticothalamic interconnections interlink widespread regions of cortex (all limbs). Corticonuclear connections travel with the corticospinal and corticobulbar systems and link sensory cortical regions with brain stem and spinal cord sensory projection nuclei and regions, providing for cortical modulation of the cortex's own sensory input. Cerebral cortex has connections with limbic forebrain structures, hypothalamic regions, and autonomic regions of the brain stem, as well as other brain stem structures (tectum, inferior olivary nucleus).

Coronal Sections through the Forebrain: Midthalamus



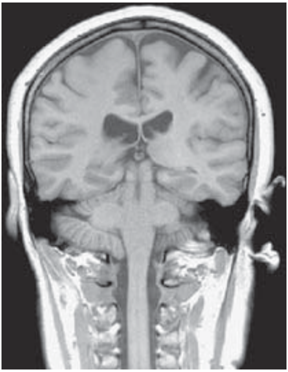


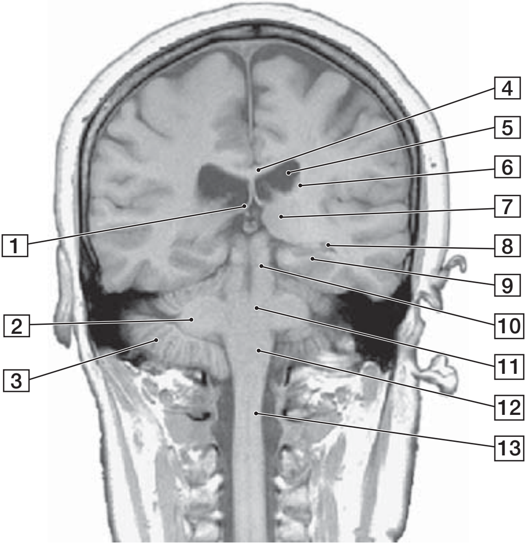
Coronal Sections through the Forebrain: Midthalamus

1. Cingulum
2. Body of the lateral ventricle
3. Columns of the fornix
4. Thalamus
5. Lateral geniculate nucleus (LGN)
6. Medial geniculate nucleus (MGN)
7. Superior cerebellar peduncle
8. Body of the caudate nucleus
9. Stria terminalis
10. Third ventricle
11. Hippocampal formation
12. Middle cerebellar peduncle
13. Inferior cerebellar peduncle

Comment: Several thalamic nuclei in the posterior thalamus convey visual and auditory information to specific regions of cerebral cortex. The LGN receives segregated (nonbinocular) input via the optic tract from the temporal hemiretina of the ipsilateral eye and nasal hemiretina of the contralateral eye (representing the contralateral visual field); the LGN further processes this information and conveys it via the geniculocalcarine pathway (visual radiations) to area 17 on the calcarine fissure banks in the occipital cortex (primary visual cortex). LGN damage can result in contralateral hemianopia. The pulvinar receives visual input from the superior colliculus (which receives visual information via the optic tract) and conveys this information to areas 18 and 19 (visual association cortex) of the occipital lobe. Damage to the pulvinar can result in contralateral visual neglect. The MGN receives input from the inferior colliculus via its brachium and from other brain stem auditory nuclei; this input represents bilateral information. Damage to the MGN does not result in unilateral (contralateral) deafness but may result in diminished hearing with contralateral neglect in response to simultaneously presented auditory stimuli.

Coronal Sections through the Forebrain: Geniculate Nuclei





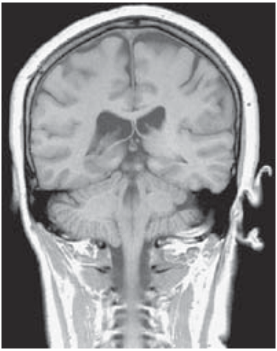
Coronal Sections through the Forebrain: Geniculate Nuclei

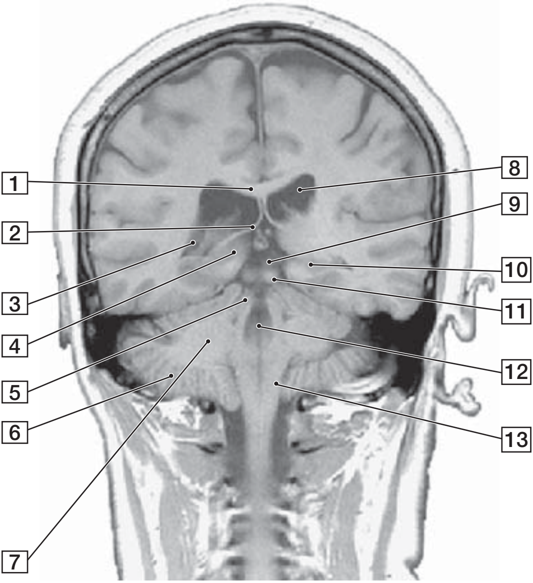
1. Columns of the fornix
2. Middle cerebellar peduncle
3. Lateral cerebellar hemisphere
4. Body of the corpus callosum
5. Body of the lateral ventricle
6. Body of the caudate nucleus
7. Pulvinar
8. Inferior horn of the lateral ventricle
9. Hippocampal formation
10. Superior cerebellar peduncle
11. Pons
12. Medulla
13. Cervical spinal cord

Comment: Cerebellar peduncles are conspicuous in coronal sections through the forebrain at the thalamus level. The inferior cerebellar peduncle conveys extensive somatosensory, trigeminal sensory, and brain stem reticular formation input to both deep nuclei and the cerebellar cortex. The cerebellum acts as a comparator, receiving extensive sensory and other input and initiating adjustments to smooth and coordinate a wide range of functions, the most conspicuous being motor movements; inebriation involves loss of cerebellar function, with resultant loss of motor coordination, as demonstrated in the cerebellar examination of field sobriety testing. The deep nuclei of the cerebellum provide the coarse adjustment, and the cerebellar provides the fine adjustment, acting through projections to deep nuclei. The middle cerebellar peduncle conveys massive input to the cerebellum through the corticopontocerebellar system, through which the cerebellum is informed of motor planning and other cortical intentions. The cerebellum output uses the superior cerebellar peduncle for connections from the deep nuclei to upper motor neuronal systems (globose and emboliform nuclei to the red nucleus, dentate nucleus to the ventral lateral nucleus of the thalamus) for the rubrospinal and corticospinal systems. The fastigial nucleus and flocculonodular lobe output travels through the inferior peduncle and helps regulate vestibulospinal and reticulospinal upper motor neuronal systems.

See book 13.18B

Coronal Sections through the Forebrain: Caudal Pulvinar and Superior Colliculus



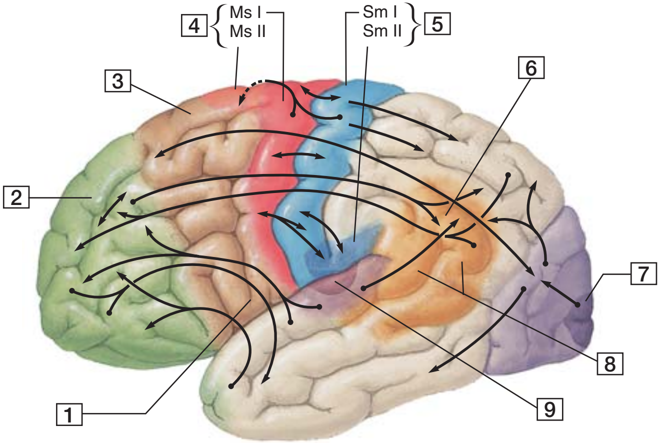


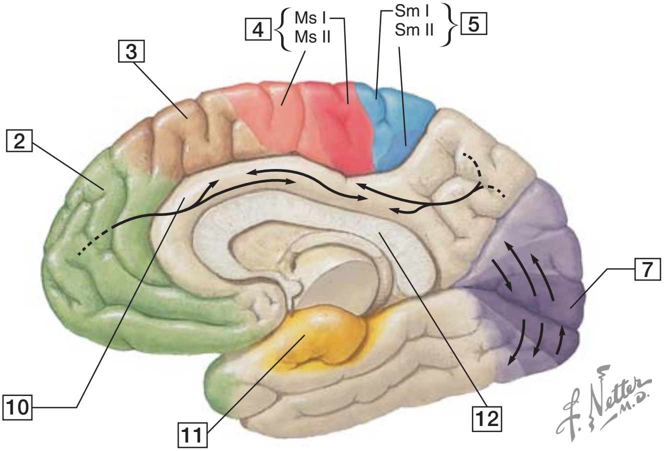
Coronal Sections through the Forebrain: Caudal Pulvinar and Superior Colliculus

1. Body of the corpus callosum
2. Columns of the fornix transitioning to the body of the fornix
3. Confluence of the inferior horn and body of the lateral ventricle
4. Pulvinar
5. Superior cerebellar peduncle
6. Lateral cerebellar hemisphere
7. Middle cerebellar peduncle
8. Body of the lateral ventricle
9. Superior colliculus
10. Hippocampal formation
11. Inferior colliculus
12. Fourth ventricle
13. Medulla

Comment: Several regions of the ventricular system are visible in coronal sections at this level. The body of the lateral ventricle and its transition to the trigone area and inferior horn are evident at this posterior region. The superior portion of the third ventricle is present medial to the columns of the fornix. The fourth ventricle and its surrounding brain structures are particularly visible in the pontine region, but the interconnecting aqueduct is present only in a more anterior coronal section. Also present superior to the superior colliculi, in more posterior coronal sections, is the cistern of the great cerebral vein, an extraventricular region of subarachnoid space. The cerebrospinal fluid system provides internal and external protection and buoyancy, preventing damage to delicate long tracts and neuronal tissue from abrupt head movements or potential rapid shearing forces.

Cortical Association Pathways



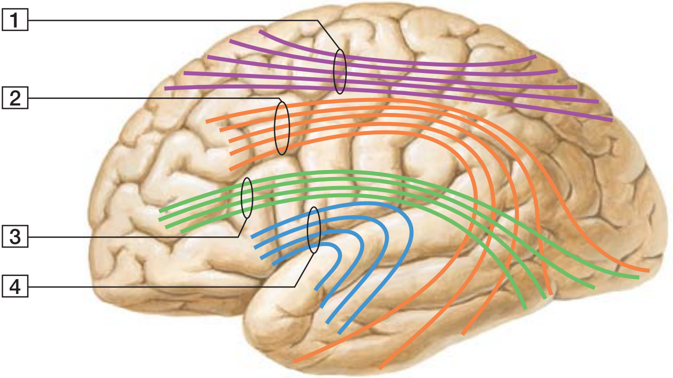


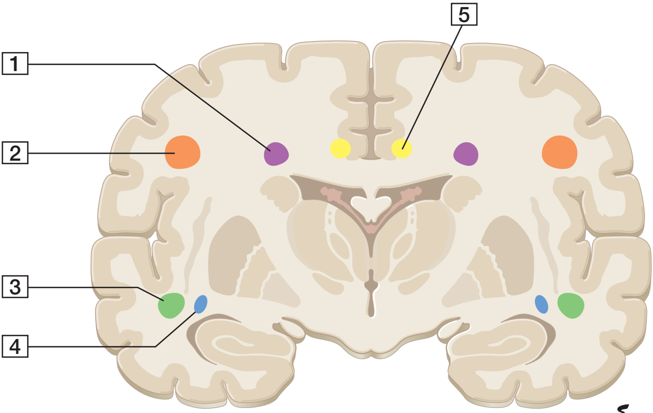
Cortical Association Pathways

1. Motor control of language and speech
2. Prefrontal cortex
3. Premotor cortex
4. Motor-sensory (Ms) cortex (primary)
5. Sensory-motor (Sm) cortex (primary)
6. Association area of sensory association areas
7. Visual cortex (I, primary)
8. Wernicke's area
9. Auditory cortex (I, primary)
10. Cingulate gyrus
11. Olfactory cortex (uncus)
12. Corpus callosum

Comment: Long cortical association pathways link regions of the cerebral cortex on one side with nonadjacent areas of the same hemisphere. Some pathways link multiple sensory cortices with association areas for multimodal processing and integrated interpretation of the outside world. Some association pathways connect language areas of the dominant hemisphere with each other. Broca's area of the frontal cortex and Wernicke's area in the parietotemporal region are interconnected by long association fibers of the arcuate fasciculus and superior longitudinal fasciculus. When these pathways are disrupted, Broca's area and Wernicke's area are dissociated; the patient does not exhibit classical expressive or receptive aphasia but demonstrates the inability to repeat complex words or sentences, a disorder called *conductive aphasia.* The cingulum is a site of multiple intercortical connections, as well as ascending and descending projection axons such as the monoaminergic axonal systems (catecholamine and serotonin); damage to these pathways can result in depression and bipolar disorder, as well as attention deficits. Bilateral damage to association pathways from the frontal lobe may result in euphoria and inappropriate affect.

Major Cortical Association Bundles





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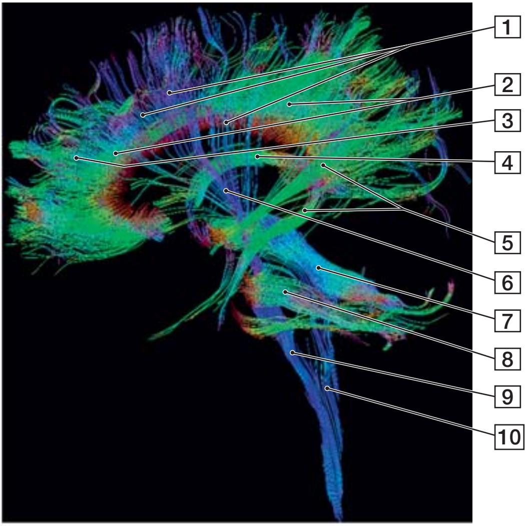
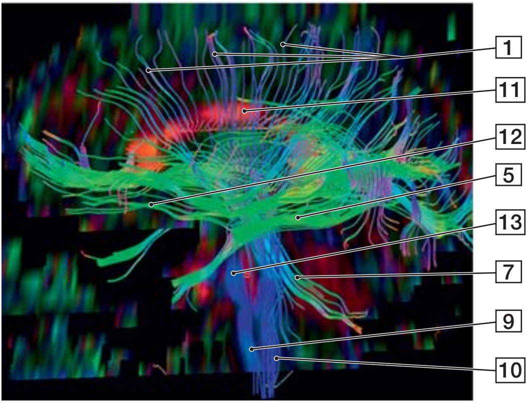
Major Cortical Association Bundles

1. Superior occipitofrontal fasciculus
2. Superior longitudinal fasciculus
3. Inferior occipitofrontal fasciculus
4. Uncinate fasciculus
5. Cingulum

Comment: Cortical association pathways or fasciculi are subject to demyelination in multiple sclerosis and other demyelinating disorders. This process can lead to cognitive and emotional impairment in addition to the specific sensory, motor, and autonomic involvement that is well known in the central demyelinating diseases. Additional problems of diminished attention and vigilance can occur with demyelination of cortical association pathways; this may contribute to some of the memory impairment seen in recall tasks. Inappropriate expression of emotion and euphoria or emotional disinhibition (sometimes called *pseudobulbar affect*) can occur with damage to frontal association pathways. Both depression and bipolar disorder occur more commonly in patients with multiple sclerosis compared with age-matched controls. Although some clinicians view some of the demyelinating plaques that form in subcortical white matter to be “silent lesions” that produce no obvious pathology, the end point for evaluation usually has been classic motor and sensory symptoms, not subtle emotional and cognitive dysfunction. Although such deficits may be more common than previously supposed, the ability of the brain to repair demyelinated lesions often can ameliorate such deficits.

Color Imaging of Projection Pathways from the Cerebral Cortex

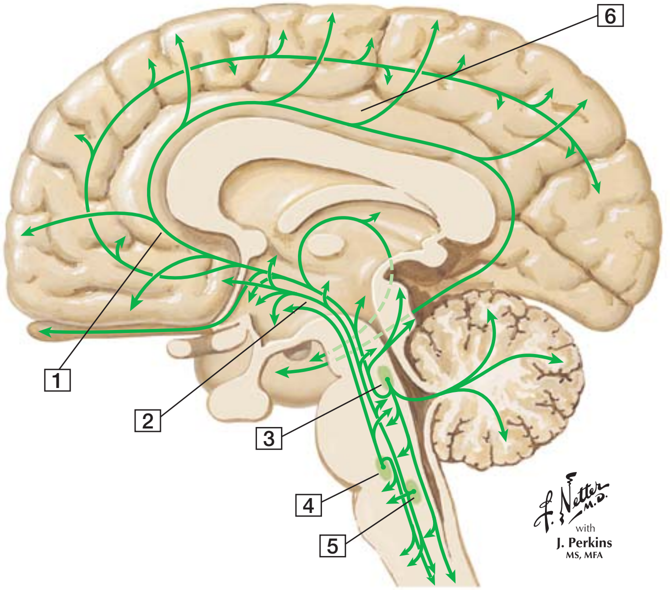




Color Imaging of Projection Pathways from the Cerebral Cortex

1. Corona radiata coalescing into the internal capsule
2. Cingulum
3. Superior longitudinal fasciculus fibers
4. Fornix
5. Inferior longitudinal fasciculus fibers
6. Internal capsule
7. Superior cerebellar peduncle
8. Middle cerebellar peduncle
9. Pyramidal tract
10. Dorsal column system
11. Corpus callosum
12. Uncinate fasciculus
13. Motor fibers in the basis pontis

Comment: Diffusion-tensor imaging (DTI), or diffusion-weighted imaging, provides pseudocolor depiction of axonal pathways and fibers on the basis of the anisotropic diffusion of water evaluated in the form of a tensor. This permits three-dimensional reconstruction of axonal pathways on the basis of eigenvectors of the diffusion tensor. In this schema, green represents association pathways, red represents commissural pathways, and blue represents projection pathways. In these images, axonal components of several long association pathways, including the cingulum, the superior longitudinal fasciculus, the inferior longitudinal fasciculus, and the uncinate fasciculus, can be identified. Commissural fibers of the corpus callosum and many projection fibers (internal capsule, fornix) and brain stem tracts (superior cerebellar peduncle, middle cerebellar peduncle, dorsal column system, corticospinal tract) also are visible with this extraordinary technique.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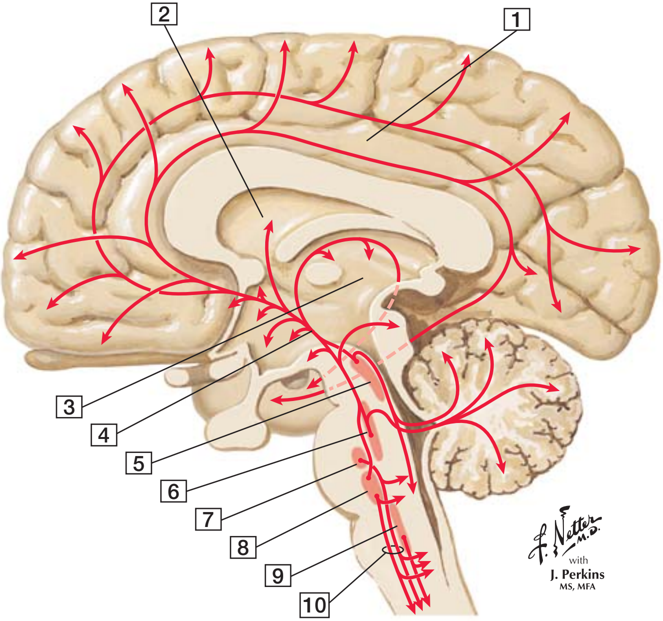
Noradrenergic Pathways

1. Dorsal noradrenergic bundle
2. Ventral noradrenergic bundle
3. Locus coeruleus (group A6)
4. Noradrenergic cell groups A5 and A7
5. Noradrenergic cell groups A1 and A2
6. Cingulum bundle

Comment: Noradrenergic (NA) neurons are located in circumscribed areas of the brain stem, labeled as “A” cell groups, most prominently found in the locus coeruleus of the pons (group A6), and in several groups in the reticular formation of the pons and medulla (groups A1, A2, A5, and A7). These neurons project to widespread regions of the central nervous system. The locus coeruleus has axons that branch to the cerebral cortex, hippocampal formation, hypothalamus, cerebellum, brain stem nuclei, and spinal cord via the dorsal NA bundle and a descending NA pathway. The locus coeruleus acts as a modulator of the excitability of other projection systems such as the glutamate system and helps to regulate attention and alertness, the sleep-wake cycle, and appropriate responses to stressors, including pain. The reticular formation groups are interconnected extensively with the spinal cord, brain stem, hypothalamic groups, and limbic regions involved in neuroendocrine control, visceral functions (temperature regulation, feeding and appetite control, drinking behavior, reproductive and sexual behavior, autonomic regulation), and emotional behavior. Serotonin neurons of the raphe system and their projections overlap with many of these NA connections and comodulate related functional activities. A sparse set of epinephrine-containing neurons (C1 and C2 groups) are found in the medullary reticular formation and modulate many brain stem functions such as blood pressure. These neurons can work in concert with the locus coeruleus during a challenge or in response to a stressor to coordinate alertness and appropriate neuroendocrine and autonomic responses. Central NA and adrenergic neuronal systems and receptors are targets of many pharmacological agents, including those for depression, analgesia, hypertension, and other conditions.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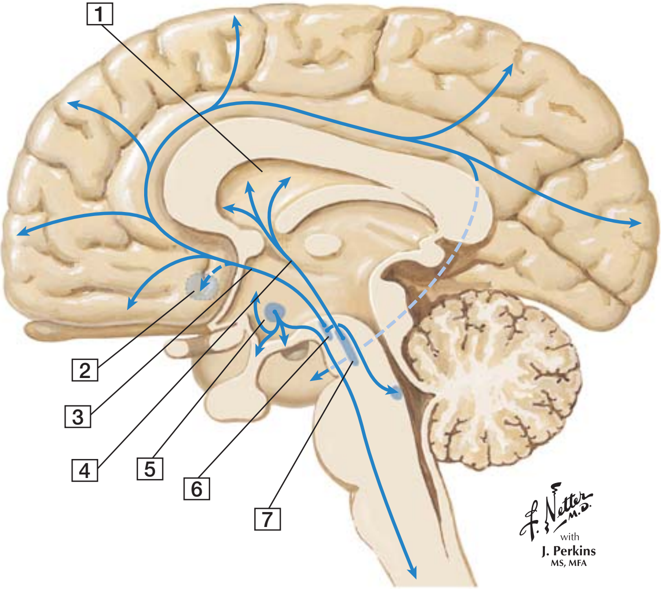
Serotonergic Pathways

1. Cingulum bundle
2. Basal ganglia
3. Thalamus
4. Ascending serotonergic pathway
5. Nucleus raphe dorsalis
6. Nucleus centralis superior
7. Nucleus raphe pontis
8. Nucleus raphe magnus
9. Nuclei raphe pallidus and obscurus
10. Descending serotonergic pathway

Comment: Serotonergic (5-hydroxytryptamine, 5-HT) neurons are found in the raphe nuclei of the brain stem and adjacent wings of cells in the reticular formation; these neurons have widespread connections that innervate every major subdivision in the CNS, providing a parallel to many, but not all, of the projections of the noradrenergic (NA) system. Rostral 5-HT neurons in the nuclei raphe dorsalis and centralis superior project rostrally to innervate the cerebral cortex, many limbic forebrain structures (hippocampal formation, amygdala), basal ganglia, many hypothalamic nuclei and areas, and some thalamic regions. Caudal 5-HT neurons in nuclei raphe pontis, magnus, pallidus, and obscurus project more caudally to innervate many regions of the brain stem, cerebellum, and spinal cord. Nucleus raphe magnus projections to the dorsal horn of the spinal cord markedly enhance opiate analgesia and alter pain processing. The ascending 5-HT systems help regulate emotional behavior and wide-ranging hypothalamic and limbic functions (neuroendocrine, visceral/autonomic), similar to their NA counterparts. 5-HT neurons are involved in sleep-wakefulness cycles and, similar to locus coeruleus NA neurons, stop firing during REM sleep. 5-HT projections to the cerebral cortex modulate the processing of afferent inputs, such as those from the visual cortex. Descending 5-HT neurons modulate preganglionic autonomic and lower motor neuron activity. Many pharmacological agents target 5-HT neurons and their receptors, including drugs for treating depression; other cognitive, emotional, and behavioral states; headaches; pain; movement disorders; and others.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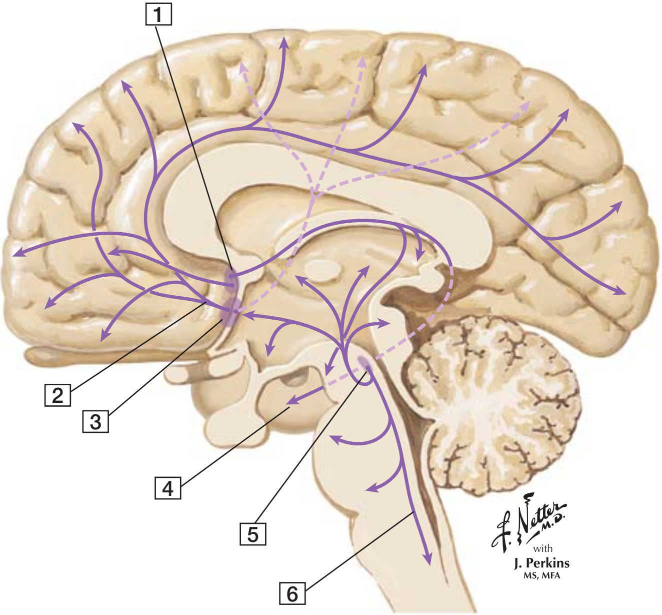
Dopaminergic Pathways

1. Striatum
2. Nucleus accumbens
3. Mesolimbic and mesocortical pathway
4. Nigrostriatal pathway
5. Hypothalamus
6. Ventral tegmental area (VTA)
7. Substantia nigra (SN), pars compacta

Comment: Dopamine (DA) neurons are found in midbrain and hypothalamus. In the midbrain, DA neurons in SN, pars compacta give rise to the nigrostriatal pathway mainly to the striatum (caudate nucleus, putamen) and, to a lesser extent, to the globus pallidus and subthalamus. Damage to the SN neurons or nigrostriatal pathway results in Parkinson's disease, characterized by resting tremor, muscular rigidity (through all ranges of motion), bradykinesia (difficulty starting or stopping movements), and postural deficits. Antiparkinson drugs such as levodopa or DA agonists target this system and its receptors. DA neurons in the VTA and mesencephalic reticular formation (group A10) send mesolimbic projections to the nucleus accumbens, amygdala, and hippocampal formation and send mesocortical projections to the frontal cortex and some cortical association areas. The mesolimbic pathway is involved in motivation, reward, biological drives, and addictive behaviors, including substance abuse. DA projections to limbic structures can induce stereotyped repetitive behaviors and activities. The mesocortical pathway influences cognitive functions in planning and carrying out frontal cortical activities and in attention mechanisms. The mesolimbic and mesocortical DA systems and receptors are targets of neuroleptic and antipsychotic agents that influence behaviors in schizophrenia, obsessive-compulsive disorder, attention deficit-hyperactivity disorder, Tourette's syndrome, and other behavioral states. DA neurons in the hypothalamus give rise to the tuberoinfundibular DA pathway, which projects from the arcuate nucleus (group A12) to the contact zone of the median eminence, where DA acts as prolactin inhibitory factor. Intrahypothalamic DA neurons (groups A11, A13) influence other neuroendocrine and visceral/autonomic hypothalamic functions.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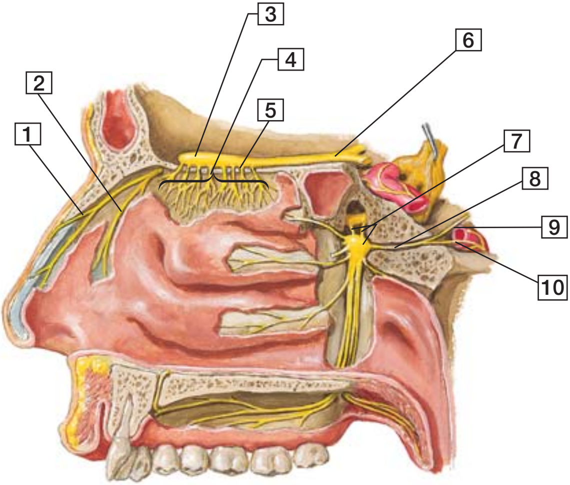
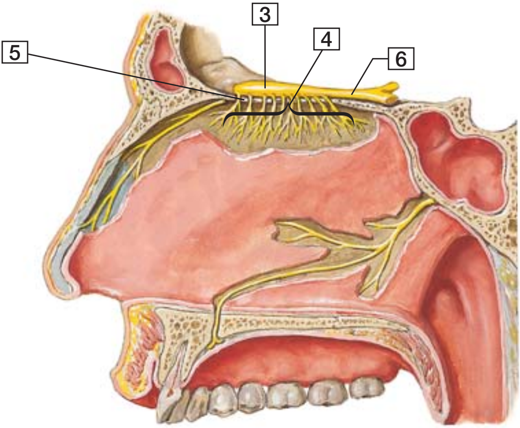
Central Cholinergic Pathways

1. Medial septal nucleus
2. Ascending cholinergic pathway
3. Nucleus basalis of Meynert
4. Hippocampus
5. Brain stem tegmental cholinergic group
6. Descending cholinergic pathway

Comment: Central cholinergic neurons are found mainly in the basal forebrain (nucleus basalis of Meynert and nucleus of the diagonal band of Broca) and in medial septal nuclei. Nucleus basalis neurons (located in substantia innominata and the ventral extent of the forebrain) project cholinergic axons to the cerebral cortex; septal cholinergic neurons project axons to the hippocampal formation. These cholinergic projections are involved in cortical activation and memory function, particularly the consolidation of short-term memory. They often appear to be damaged in patients with Alzheimer's disease; drugs that enhance cholinergic function are used for attempted improvement of memory. Other cholinergic neurons found in the brain stem tegmentum project to structures in the thalamus, brain stem, and cerebellum; the projections to the thalamus modulate arousal and the sleep-wake cycle and appear to be important in the initiation of REM sleep. Cholinergic interneurons are present in the striatum and may participate in basal ganglia control of ton, posture, and initiation of movement or selection of wanted patterns of activity. In some instances, drugs aimed at reducing cholinergic activity are used in Parkinson's disease and a complementary or alternate approach to levodopa or DA agonists. Acetylcholine is also used as the principal neurotransmitter in all preganglionic autonomic neurons, as well as in all lower motor neurons of the spinal cord and brain stem.

Olfactory Nerves





Olfactory Nerves

1. Anterior ethmoidal nerve, external branch
2. Anterior ethmoidal nerve, internal nasal branch
3. Olfactory bulb
4. Olfactory nerves
5. Cribriform plate of the ethmoid bone
6. Olfactory tract
7. Pterygopalatine ganglion and branches
8. Nerve of the pterygoid canal
9. Greater petrosal nerve
10. Deep petrosal nerve

Comment: The olfactory nerves (CN I) and their projections into the CNS are important components of forebrain function. Bipolar cells in the olfactory epithelium are the primary sensory neurons. Their peripheral (CNS) axons act as a chemosensory transducer and respond to unique chemical stimuli of airborne molecules entering the nose (responsiveness to at least 10,000 odors). Central axons of the bipolar neurons aggregate into groups of approximately 20 slender olfactory nerves that traverse the cribriform plate and terminate in glomeruli of the ipsilateral olfactory bulb. These nerves are vulnerable to tearing at the cribriform plate, resulting in anosmia. Olfactory bipolar neurons can regenerate and proliferate even though they are CNS neurons. After processing of information in the olfactory bulb, mitral neurons and tufted neurons project via the olfactory tract directly and indirectly to limbic forebrain structures, including septal and amygdaloid nuclei. These projections bypass the thalamus, have immediate access to limbic forebrain structures, and directly influence the hypothalamus and its regulation of neuroendocrine and visceral/autonomic functions, including appetite, nausea, relaxation and sleep, alertness, sexual arousal, and others. The olfactory system is essential to survival in many species in territorial recognition, defense, food and water acquisition, social and reproductive behavior, and danger signaling.

3

Systemic Neuroscience

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3

Systemic Neuroscience
Plates 3-1 to 3-59

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Systemic Neuroscience

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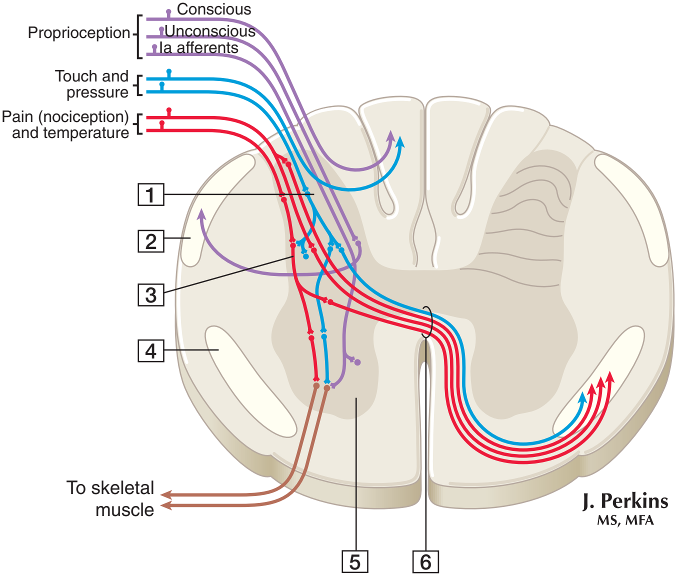
Somatosensory Afferents to the Spinal Cord

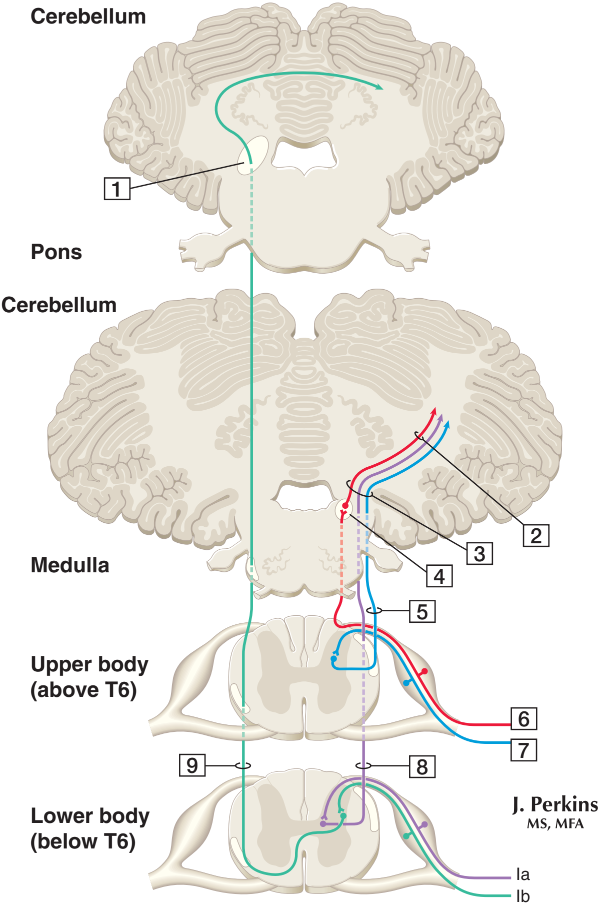


Somatosensory Afferents to the Spinal Cord

1. Dorsal horn
2. Dorsal spinocerebellar tract
3. Interneurons for spinoreticular nociceptive system
4. Spinothalamic/spinoreticular tracts (anterolateral system)
5. Ventral horn—lower motor neurons
6. Anterior white commissure

Comment: Epicritic afferents (mainly larger-diameter myelinated axons) convey fine discriminative touch, vibratory sensation, joint position sense, and secondary modalities such as graphesthesia and two-point discrimination. Protopathic afferents (mainly small myelinated and unmyelinated axons) convey mainly nociceptive information and temperature sensation. Epicritic afferents processing modalities for conscious interpretation enter the spinal cord at the dorsal root entry zone and turn rostrally in fasciculi gracilis (T6 and below) and cuneatus (above T6), terminating in nuclei gracilis and cuneatus in the medulla. Primary afferents destined for spinocerebellar inputs (mainly myelinated) enter the spinal cord and terminate in secondary sensory nuclei in the spinal cord or caudal medulla; secondary sensory projections send information to the ipsilateral cerebellum. Protopathic axons of two categories enter the spinal cord. Small myelinated axons and some unmyelinated axons conveying “fast” pain and temperature sensation (localized and not outlasting the duration of the stimulus) terminate in lamina I and V of the dorsal horn; secondary sensory projections cross into the spinothalamic tract and terminate in the thalamus (ventral posterolateral nucleus, VPL). Small unmyelinated axons conveying prolonged, excruciating pain that outlasts the duration of the stimulus terminate on interneurons in the dorsal horn, which connect with a cascade of neurons that contribute to the bilateral polysynaptic spinoreticular system that terminates in the reticular formation of the brain stem and is then conveyed through the nonspecific thalamus.

Somatosensory System: Spinocerebellar Pathways

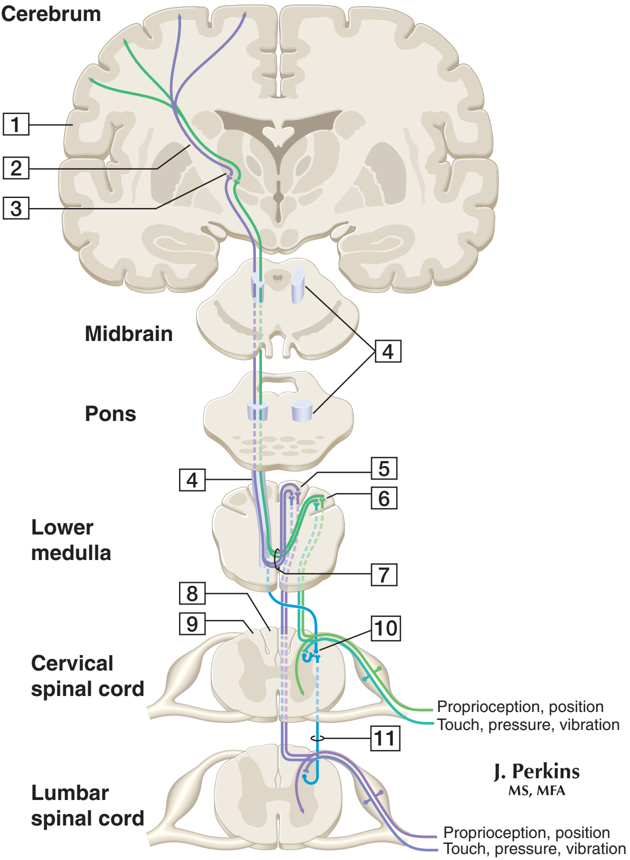


Somatosensory System: Spinocerebellar Pathways

1. Superior cerebellar peduncle
2. Cuneocerebellar tract
3. Inferior cerebellar peduncle
4. Lateral (accessory) cuneate nucleus
5. Rostral spinocerebellar tract (RSCT)
6. Ia primary afferent
7. Ib primary afferent
8. Dorsal spinocerebellar tract
9. Ventral spinocerebellar tract (VSCT)

Comment: Proprioceptive primary sensory axons from the joints, tendons, and ligaments (represented in diagram by Ib afferents) terminate on secondary sensory neurons (border cells, dorsal horn neurons) of the ventral spinocerebellar tract and rostral spinocerebellar tract, carrying information from the lower (T6 and below) and upper (above T6) body, respectively. Proprioceptive primary somatosensory axons from muscle spindles (represented in diagram by Ia afferents) terminate on secondary sensory neurons (Clarke's nucleus and the lateral [external] cuneate nucleus) of the dorsal spinocerebellar tract and cuneocerebellar tract, carrying information from the lower (T6 and below) and upper (above T6) body, respectively. The spinocerebellar tracts terminate ipsilaterally in the cerebellar except for the ventral spinocerebellar tract, which crosses once in the spinal cord through the anterior white commissure, and then again in the cerebellum, effectively conveying information to the ipsilateral cerebellum. The output of the cerebellum also is conveyed, sometimes through two sets of crossing fibers, back to the ipsilateral side. Thus, cerebellar lesions generally result in ipsilateral symptoms.

Somatosensory System: Dorsal Column System and Epicritic Modalities

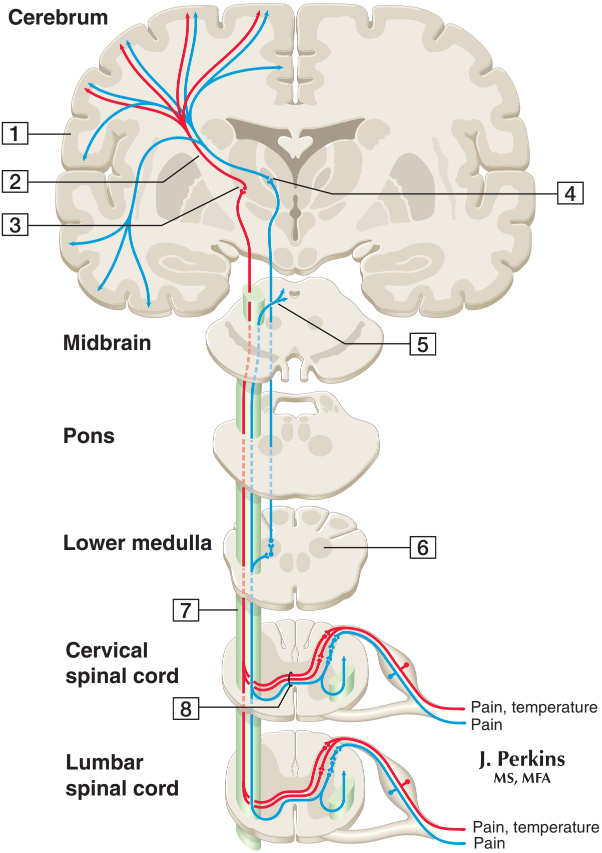


Somatosensory System: Dorsal Column System and Epicritic Modalities

1. Cerebral cortex: postcentral gyrus
2. Posterior limb of the internal capsule
3. Ventral posterolateral (VPL) nucleus of the thalamus
4. Medial lemniscus
5. Nucleus gracilis
6. Nucleus cuneatus
7. Internal arcuate fibers
8. Fasciculus gracilis
9. Fasciculus cuneatus
10. Lateral cervical nucleus (C1 and C2 only)
11. Spinocervical tract

Comment: The dorsal column consists of the fasciculi gracilis (T6 and below) and cuneatus (above T6) which convey epicritic information fine discriminative touch, vibratory sensation, joint-position sense, and secondary somatosensory modalities in myelinated axons to nuclei gracilis and cuneatus. The secondary sensory axons of the medial lemniscus are crossed (internal arcuate fibers) and terminate in VPL nucleus of the thalamus. This nucleus projects to the primary somatosensory cortex on the postcentral gyrus of the parietal lobe, as well as to secondary somatosensory cortex. Thus, epicritic information is interpreted in the contralateral hemisphere. Supplemental epicritic information also is conveyed through the dorsal portion of the lateral funiculus through the dorsal horn and the dorsal column nuclei. This supplemental information accounts for the remaining presence of some vibratory sensation and other epicritic information in patients with extensive or even complete dorsal column lesions. Lesions in the spinal cord may affect epicritic fibers or protopathic fibers selectively, producing dissociated sensory loss of those modalities without producing full anesthesia; selective lesions of the peripheral nerves may also produce selective dissociated sensory loss by involving small-diameter unmyelinated fibers (leprosy) or large-diameter unmyelinated fibers (Guillain-Barré syndrome).

Somatosensory System: Spinothalamic and Spinoreticular Systems and Protopathic Modalities

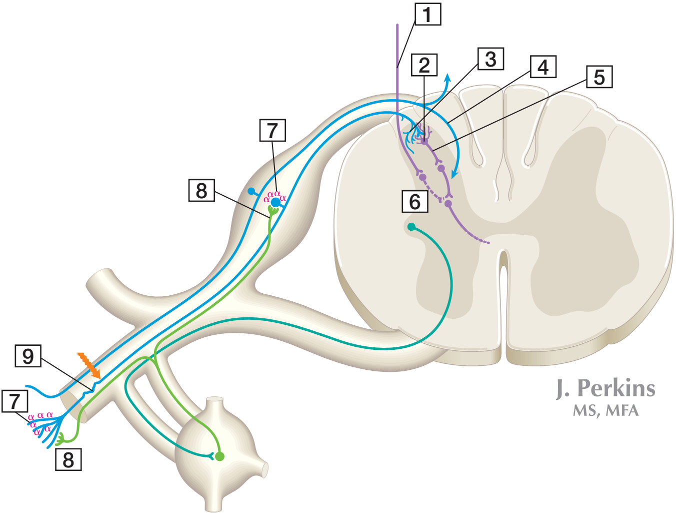


Somatosensory System: Spinothalamic and Spinoreticular Systems and Protopathic Modalities

1. Cerebral cortex (postcentral gyrus)
2. Posterior limb of the internal capsule
3. Ventral posterolateral (VPL) nucleus of the thalamus
4. Nonspecific thalamic nuclei
5. Deep layers of superior colliculus, periaqueductal gray
6. Lateral reticular formation
7. Spinothalamic/spinoreticular system
8. Anterior white commissure

Comment: The spinothalamic tract conveys lemniscal information from primary afferents for nociception and temperature sensation to secondary sensory nuclei in lamina I and V of the dorsal horn; these neurons give rise to the spinothalamic tract, which terminates in the contralateral VPL nucleus of the thalamus. This "fast pain" and temperature, which do not outlast the duration of the stimulus, are localizing and are processed in the contralateral hemisphere (primary and secondary somatosensory cortex). This is the system tested with pin prick and temperature testing in the neurological examination. Chronic, agonizing "deep" pain is processed through a cascading assembly of neurons in the dorsal horn of the spinal cord, conveyed bilaterally to the reticular formation through the spinoreticular system, and then further diffusely processed through brain stem structures, nonspecific thalamic nuclei, and limbic and cortical structures for more subjective aspects of chronic pain. This spinoreticular system can be "gated" by inputs from the dorsal column nuclei, from brain stem monoaminergic nuclei, from opioid peptide systems of the brain stem and hypothalamus, and from limbic and cortical structures.

Mechanisms of Neuropathic Pain and Sympathetically Maintained Pain

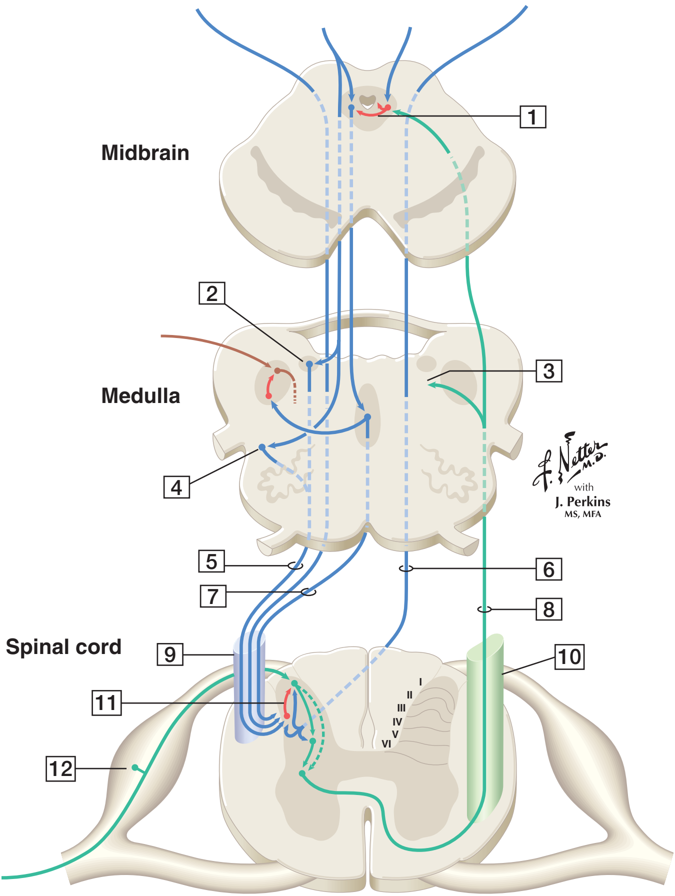


Mechanisms of Neuropathic Pain and Sympathetically Maintained Pain

1. Central serotonin and central norepinephrine pathways
2. Extension of interneuron dendrites into adjacent laminae
3. Sprouting of C fibers in the spinal cord
4. Immobilization by pain decreasing gating of nociceptive input
5. Permanent hyperactivation of wide, dynamic-range neurons
6. Glutamate excitotoxic cell death of inhibitory neurons
7. Proliferation of α-adrenergic receptors on primary sensory afferent endings and cell bodies
8. Sprouting of sympathetic postganglionic nerve fibers on primary afferent endings and primary sensory cell bodies
9. Lowered threshold for firing of C fibers (hyperesthesia) and Aδ fibers (allodynia)

Comment: In a small percent of cases of nerve damage or compression (e.g., sprain, crush injury, minor trauma), a reaction of primary afferents and then central neural structures can result in a chronic neuropathic pain syndrome called *complex regional pain syndrome* (CRPS), formerly called *reflex sympathetic dystrophy* (RSD). The pain is chronic, intense, burning, or stabbing, and often agonizing; such pain affects the hand, arm, and shoulder more than the lower extremity. The patient often reports allodynia (extreme sensitivity to touch) and hyperesthesia (extreme sensitivity to painful stimuli). Mechanisms noted in 7 and 8 earlier can provoke extraordinary sensitivity to catecholamines, lowering the threshold for response to nociceptive stimuli. These sympathetically related characteristics may produce vasomotor changes (vascular flow changes), trophic changes (atrophic skin and nails), pseudomotor changes (altered sweating and skin temperature), and altered bone density on a triphasic bone scan. Multimodal pharmacological therapy and nerve or dorsal column stimulation often are employed as treatment. Treatment must be provided quickly after detection because this disorder may spread centrally, become bilateral, and become intractable to treatment.

Descending Control of Pain Processing

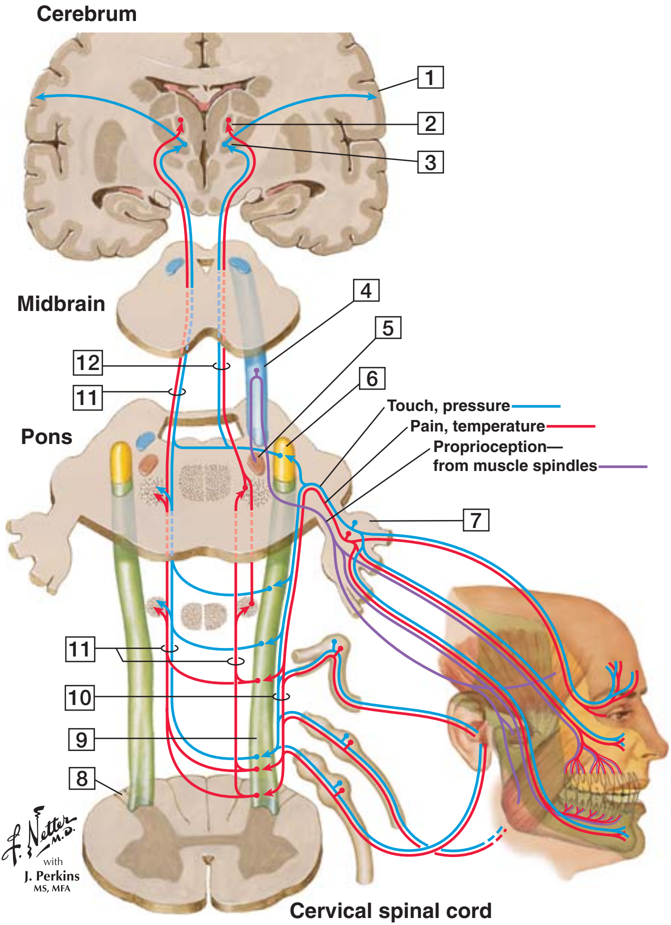


Descending Control of Pain Processing

1. Enkephalin-containing neuron in periaqueductal gray matter
2. Locus coeruleus
3. Lateral reticular formation
4. Brain stem tegmental noradrenergic cell groups
5. Descending norepinephrine pathway
6. Corticonuclear fiber
7. Descending serotonin pathway
8. Spinoreticular pathway
9. Posterolateral funiculus
10. Anterolateral funiculus
11. Enkephalin-containing neuron
12. Afferent pain neuron of dorsal root ganglion

Comment: Nociceptive information processing in the dorsal horn of the spinal cord, associated with the spinoreticular system, can be modulated by descending connections from the cerebral cortex (corticonuclear), limbic forebrain structures, the hypothalamus (such as the paraventricular nucleus and the periarcuate beta-endorphin neurons), the periaqueductal gray, the brain stem reticular formation, central noradrenergic neurons (locus coeruleus, brain stem tegmental groups), central serotonergic neurons (especially nucleus raphe magnus), and primary afferent collaterals from the dorsal column system. The central descending noradrenergic and serotonergic pathways, modulated by the periaqueductal gray and other higher centers, are particularly important for endogenous and exogenous opioid modulation of pain. Synthetic opiates may activate neurons of the periarcuate region of the hypothalamus and periaqueductal gray, enhancing analgesia. These descending pathways also can modulate trigeminal pain through the descending nucleus of V.

Trigeminal Sensory and Associated Sensory Systems

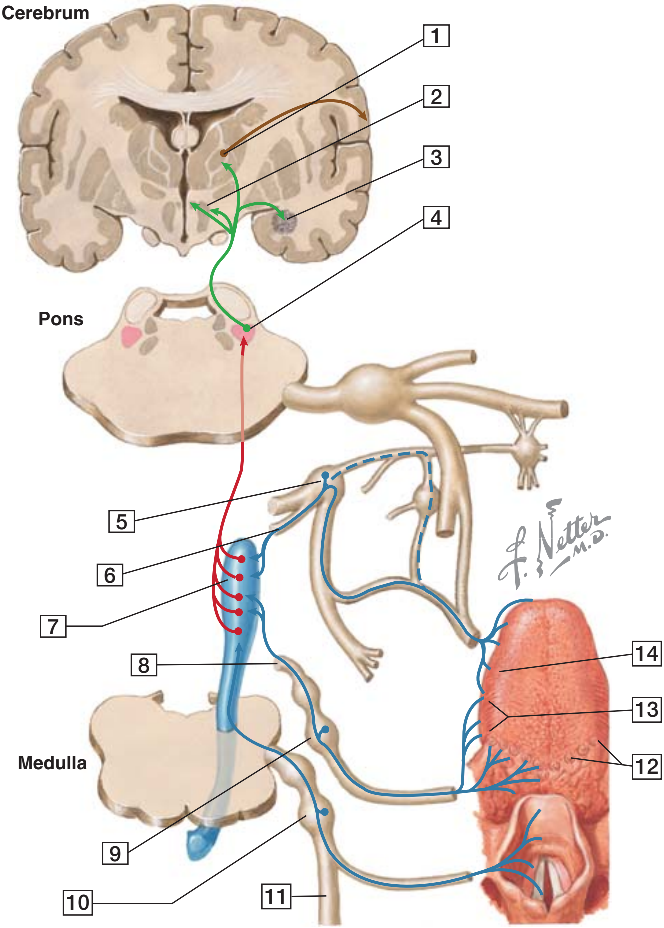


Trigeminal Sensory and Associated Sensory Systems

1. Cerebral cortex: postcentral gyrus
2. Centromedian nucleus of the thalamus (intralaminar)
3. Ventral posteromedial (VPM) nucleus of the thalamus
4. Trigeminal mesencephalic nucleus
5. Trigeminal motor nucleus
6. Principal (main) sensory trigeminal nucleus
7. Trigeminal (semilunar) ganglion
8. Dorsolateral fasciculus (of Lissauer)
9. Spinal (descending) trigeminal nucleus
10. Spinal (descending) trigeminal tract
11. Ventral trigeminal lemniscus (ventral trigeminothalamic tract)
12. Dorsal trigeminal lemniscus (dorsal trigeminothalamic tract)

Comment: Primary sensory trigeminal axons convey protopathic information (nociceptive, temperature sensation, light moving touch) into the descending tract of V, and terminate in the descending nucleus of V. The trigeminal ganglion conveys information to this nucleus from the face, anterior oral cavity, and teeth and gums; axons of the geniculate ganglion (VII) and jugular ganglion (X) supply a small zone of the external ear. Axons of the petrosal ganglion (IX) convey sensation from the posterior oral cavity and pharynx to the descending nucleus of V. The descending nucleus of V gives rise to the crossed ventral trigeminal lemniscus, which terminates in nucleus VPM of the thalamus, which projects to the lateral portion of the postcentral gyrus. Portions of the descending nucleus of V project to the reticular formation, conveying "slow" agonizing pain, similar to the reticulospinal system. Primary trigeminal axons conveying epicritic (fine, discriminative touch) information, similar to the dorsal column system, terminate in the rostral portion of the descending nucleus of V and the main sensory nucleus. Although a portion of the main sensory nucleus of V projects ipsilaterally, most of the epicritic processing takes place in the contralateral hemisphere, similar to the somatosensory system. Sensations of taste and associated trigeminal sensation are conveyed mainly ipsilaterally.

Taste Pathways



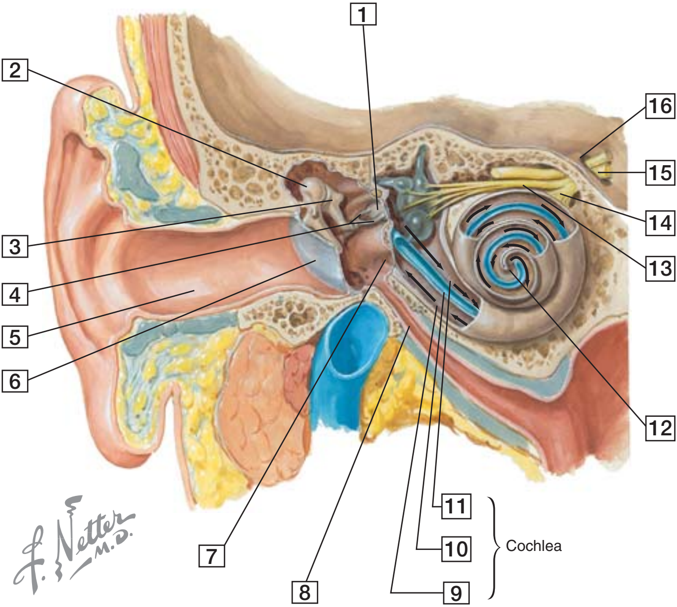
Taste Pathways

1. Ventral posteromedial (VPM) nucleus of the thalamus
2. Lateral hypothalamic area
3. Amygdala
4. Pontine taste area (parabrachial nucleus)
5. Geniculate ganglion
6. Nervus intermedius
7. Rostral part of nucleus of the solitary tract
8. Glossopharyngeal (IX) nerve
9. Petrosal (inferior) ganglion of CN IX
10. Nodose (inferior) ganglion of CN X
11. Vagus nerve (CN X)
12. Vallate papillae
13. Foliate papillae
14. Fungiform papillae

Comment: Primary sensory axons of neurons in the geniculate ganglion (VII), petrosal ganglion (IX), and nodose ganglion (X) supply taste buds on the anterior two thirds of the tongue, posterior one third of the tongue, and epiglottis and palate, respectively. These axons terminate in the rostral part of nucleus solitarius, which projects mainly to the ipsilateral parabrachial nucleus in the pons. The parabrachial nucleus conveys taste information to the ipsilateral nucleus VPM of the thalamus, several hypothalamic sites, and the amygdaloid complex. Some cortical areas, such as the anterior insular cortex and the posterior orbitofrontal cortex, are involved in the subjective, motivational, emotional, and behavioral aspects of taste and gustatory experience. Taste sensation may be blunted by a cold, many pharmaceutical agents, chemotherapy, alterations in olfactory processing, and many illnesses.

Peripheral Pathways for Sound Reception

Frontal section

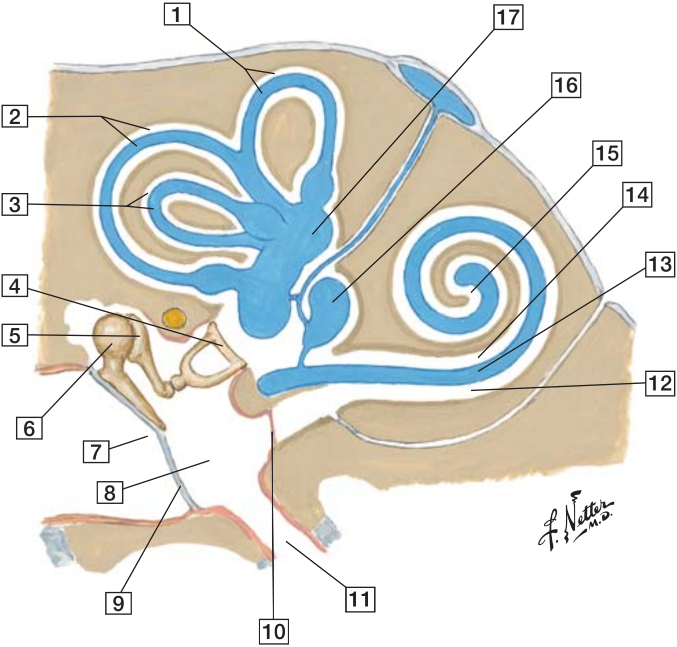


Peripheral Pathways for Sound Reception

1.	Base of stapes in oval window
2.	Malleus
3.	Incus
4.	Limbs of stapes
5.	External acoustic meatus
6.	Tympanic membrane
7.	Round (cochlear) window
8.	Pharyngotympanic (auditory) tube
9.	Scala tympani
10.	Cochlear duct containing spiral organ of Corti
11.	Scala vestibuli
12.	Helicotrema
13.	Vestibular nerve
14.	Cochlear nerve
15.	Vestibulocochlear nerve (CN VIII)
16.	Internal acoustic meatus

Comment: Sound transduction is a complex process starting with sound waves channeled through the external ear and external acoustic meatus to the tympanic membrane. Movement of this membrane is leveraged mechanically by the ossicles of the middle ear (malleus, incus, stapes) against the oval window, producing a fluid wave in the cochlear duct. This fluid wave causes differential movement of the basilar membrane, on which the hair cells of the organ of Corti are located, against the tectorial membrane above the hair cells. Stimulation of the hairs on the apical portion of the hair cells results in the release of neurotransmitters that stimulate the primary afferents of neurons of the spiral ganglion. The basilar membrane in the cochlea shows maximal displacement spatially according to the frequency of the impinging tone; low frequencies maximally stimulate the apex (helicotrema), while high frequencies maximally stimulate the base. The pharyngotympanic tube permits pressure equalization between the middle ear and the outside world. Hearing loss can involve either a conduction deficit (through damage to the transduction process) or sensorineural loss (nerve related). Use of the Weber test and the Rinne test can assist in distinguishing these types of hearing loss.

Bony and Membranous Labyrinths

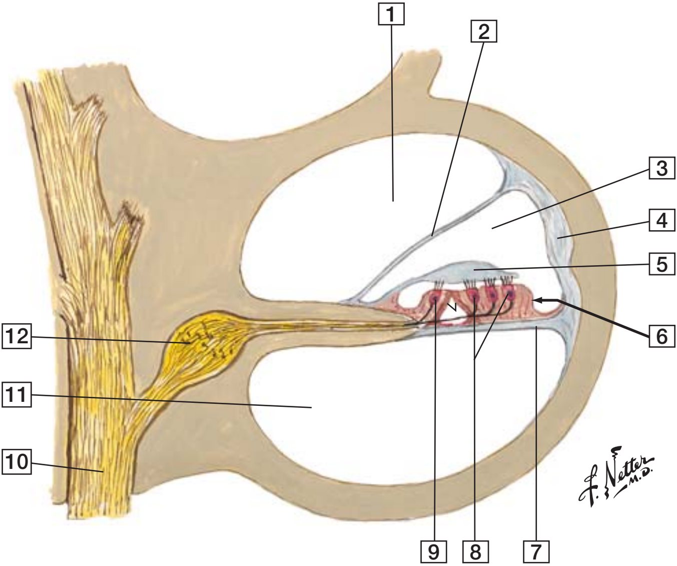


Bony and Membranous Labyrinths

1. Anterior semicircular canal and duct
2. Posterior semicircular canal and duct
3. Lateral semicircular canal and duct
4. Stapes in oval window
5. Incus
6. Malleus
7. External acoustic meatus
8. Tympanic cavity
9. Tympanic membrane
10. Round window
11. Pharyngotympanic (auditory) tube
12. Scala tympani
13. Cochlear duct
14. Scala vestibuli
15. Helicotrema of cochlea
16. Saccule
17. Utricle

Comment: The semicircular canals (ducts) contain the ampullae with hair cells that respond to angular acceleration. The utricle contains the otolith organ in the macula that responds to linear acceleration and detects gravitation. The saccule responds best to vibratory stimuli. The cochlea contains the hair cells that respond to fluid movements within the scala vestibule and tympani; these affect fluid movement in the cochlear duct and are brought about by mechanical leveraging of the ossicles against the oval window. The activity of the utricle can sometimes become distorted when debris moves away from the hairs and induces activation of the hair cells in the ampulla of the posterior semicircular canal. This produces vertigo and nystagmus that are associated with a specific position of the head, known as *benign postural* or *positional vertigo.*

VIII Nerve Innervation of Hair Cells of the Organ of Corti

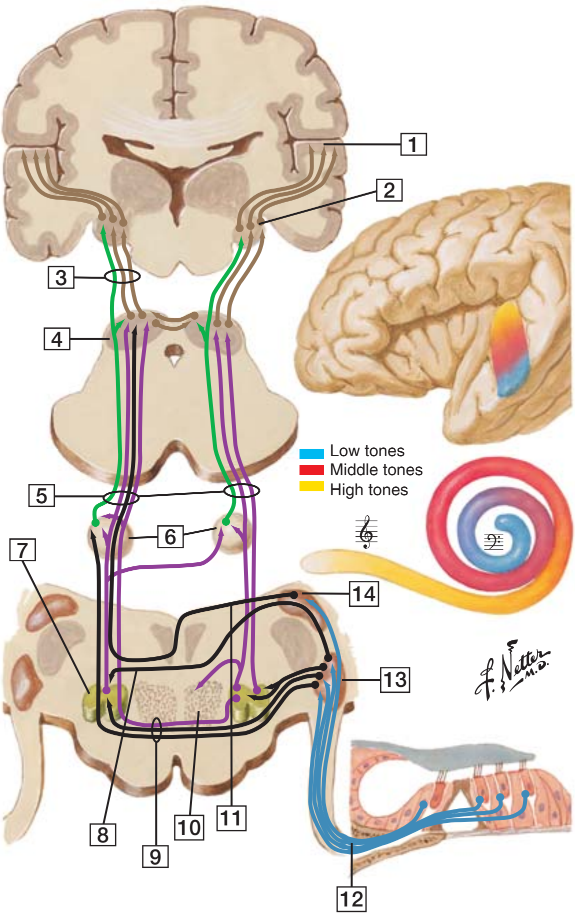


VIII Nerve Innervation of Hair Cells of the Organ of Corti

1. Scala vestibuli
2. Vestibular (Reissner's) membrane
3. Cochlear duct
4. Spiral ligament
5. Tectorial membrane
6. Spiral organ of Corti
7. Basilar membrane
8. Outer hair cells
9. Inner hair cells
10. Cochlear nerve
11. Scala tympani
12. Spiral ganglion

Comment: Primary sensory axons of the spiral (cochlear) ganglion innervate inner and outer hair cells in the organ of Corti, located on the basilar membrane. The axons are activated by neurotransmitters released from the hair cells, induced by shearing forces on the hairs at the apical surface of the hair cells due to displacement of the basilar membrane by fluid wave motion through the cochlear duct, in relation to the more rigidly fixed tectorial membrane above. This represents the complex transduction process converting mechanical movement from sound waves into action potentials in the axons of spiral ganglion neurons. Potential differences from ion composition in the scala tympani (perilymph, 0 mv) and scala vestibuli (perilymph, weakly + mv) versus the cochlear duct (endolymph, +80 mv) contribute to the excitability of the hair cells.

Auditory Pathways

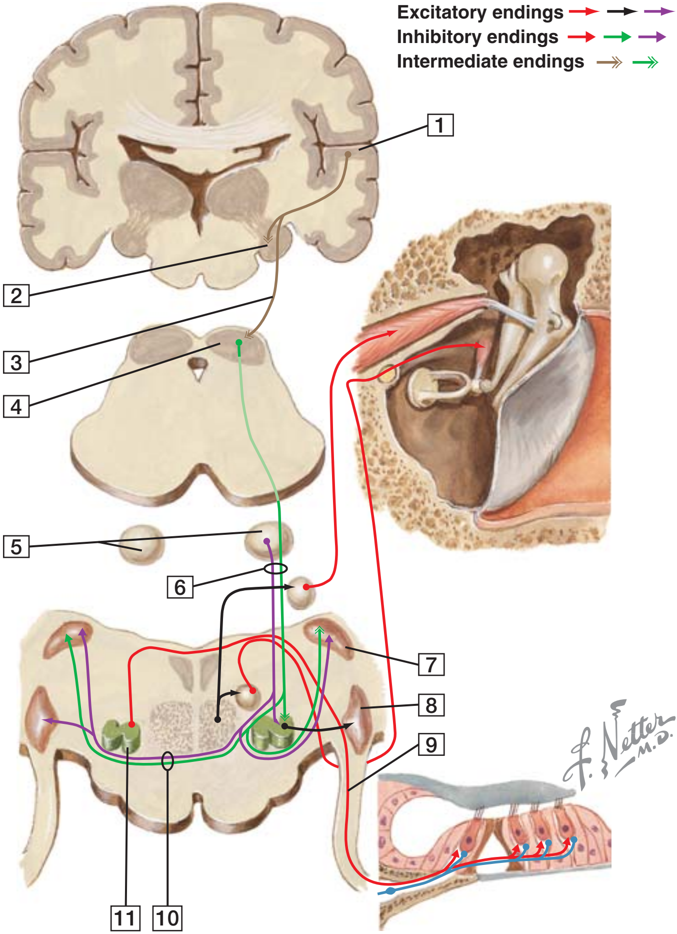


Auditory Pathways

1. Acoustic area of the temporal lobe cortex
2. Medial geniculate nucleus (body)
3. Brachium of the inferior colliculus
4. Inferior colliculus
5. Lateral lemnisci
6. Nuclei of the lateral lemnisci
7. Superior olivary complex
8. Intermediate acoustic stria
9. Trapezoid body (ventral acoustic stria)
10. Reticular formation
11. Dorsal acoustic stria
12. Spiral ganglion
13. Ventral cochlear nucleus
14. Dorsal cochlear nucleus

Comment: The cochlear nerve contains axons that innervate the hair cells of the organ of Corti in the spirals of the cochlea. Central projections of these axons terminate in the dorsal and ventral cochlear nuclei in several tonotopically organized maps. The cochlear nuclei then project into the lateral lemniscus via the acoustic stria; these projections are bilateral. The lateral lemniscus terminates in the nucleus of the inferior colliculus, which in turn projects via the brachium of the inferior colliculus to the medical geniculate nucleus in the thalamus. This thalamic nucleus reciprocally interconnects with the primary auditory cortex on the transverse gyrus of Heschl in the temporal lobe near the lateral fissure. Several additional accessory auditory nuclei receive bilateral projections from the main auditory pathways and in turn project bilaterally. Thus, sound is represented throughout the afferent pathways bilaterally; a unilateral lesion in a site rostral to the cochlear nuclei results in diminished hearing, not a unilateral hearing loss. Damage to the auditory radiations or auditory cortex results in a diminution in hearing and auditory neglect contralateral to the lesion during bilateral simultaneous presentation of sound.

Centrifugal (Efferent) Auditory Pathways



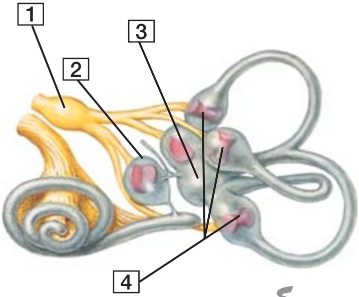
Centrifugal (Efferent) Auditory Pathways

1. Temporal cortex
2. Medial geniculate nucleus (body)
3. Brachium of the inferior colliculus
4. Inferior colliculus
5. Nuclei of the lateral lemnisci
6. Lateral lemnisci
7. Dorsal cochlear nucleus
8. Ventral cochlear nucleus
9. Efferent olivocochlear fibers
10. Trapezoid body
11. Superior olivary complex

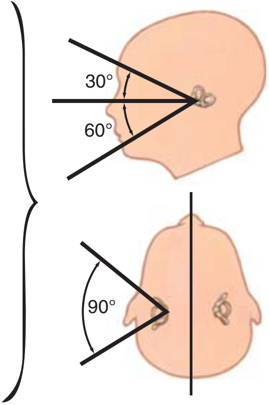
Comment: Descending pathways travel from the auditory cortex, the medial geniculate nucleus, the inferior colliculus, and accessory auditory nuclei in the brain stem to terminate in caudal structures in the auditory pathways, such as the cochlear nuclei and the superior olivary nuclei. These centrifugal connections permit descending control of incoming auditory information. The olivocochlear bundle from the superior olivary nuclei projects back to the hair cells in the organ of Corti and modulates the transduction process between the hair cells and the primary afferent axons. These centrifugal connections permit higher auditory processing zones to focus on specific inputs, providing the ability to tune into a specific conversation or auditory stimulus in the midst of extensive auditory background information, such as listening to one distant conversation at a cocktail party.

Vestibular Receptors

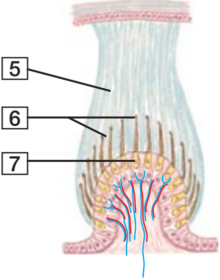
A. Membranous labyrinth



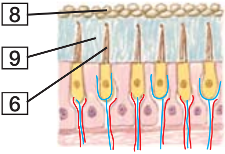
B. Position within base of skull



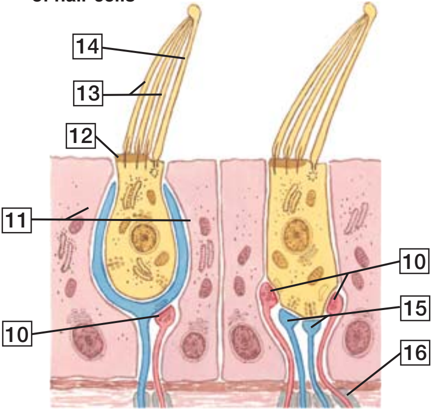
C. Section of crista



D. Section of macula



E. Structure and innervation of hair cells

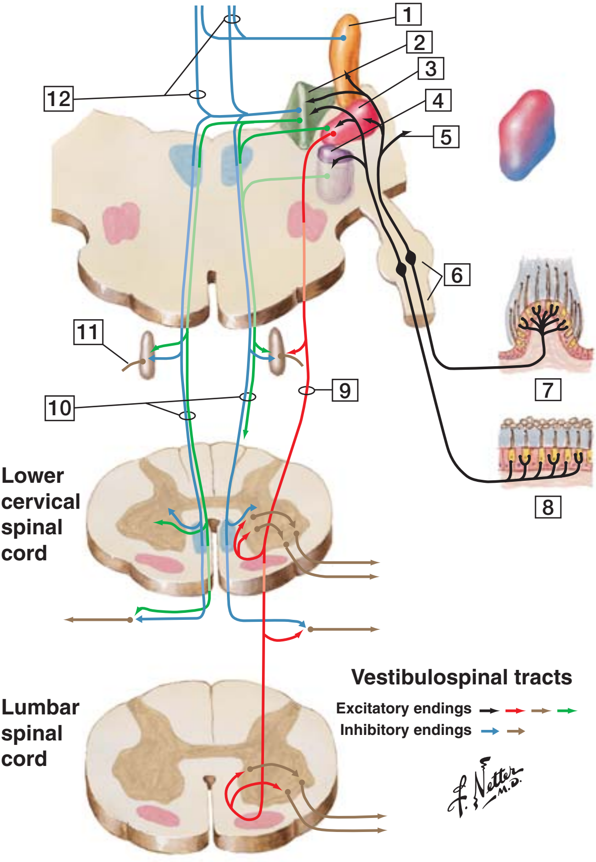


Vestibular Receptors

1. Vestibular ganglion
2. Saccule
3. Utricle
4. Cristae within ampullae
5. Gelatinous cupula
6. Hair tufts
7. Hair cells
8. Otoconia
9. Gelatinous otolith membrane
10. Efferent nerve ending
11. Supporting cells
12. Cuticle
13. Stereocilia
14. Kinocilium
15. Afferent nerve calyx
16. Myelin sheath

Comment: The vestibular receptors include hair cells in the cristae ampullaris of the utricle (linear acceleration, gravity) and saccule (low frequency vibration) and in the maculae of the orthogonally oriented semicircular canals (angular acceleration, movement). Hair tufts from the cristae ampullaris and the maculae are embedded in a gelatinous substance that responds to gravity (utricle, otolith forces) or head movement (kinocilium movement), depolarizing the hair cell and resulting in release of a neurotransmitter that stimulates the associated primary afferent of the vestibular (Scarpa's) ganglion. This system also has centrifugal or efferent modulation of the primary afferent transduction process and associated afferent pathways.

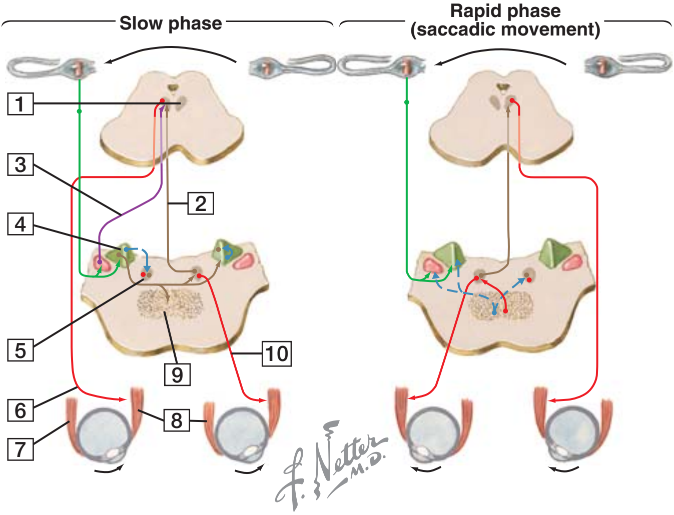
Vestibular Pathways



Vestibular Pathways

1. Superior vestibular nucleus
2. Medial vestibular nucleus
3. Lateral vestibular nucleus
4. Inferior vestibular nucleus
5. Primary vestibular afferent fibers to the cerebellum
6. Vestibular ganglion and nerve
7. Fibers from cristae ampullaris
8. Fibers from maculae
9. Lateral vestibulospinal tract
10. Medial vestibulospinal fibers in the medial longitudinal fasciculus
11. Motor neuron controlling neck musculature
12. Ascending fibers in medial longitudinal fasciculus

Comment: The vestibular nerve consists of primary afferents from the cristae in the ampullae of the semicircular canals and from the maculae of the utricle and saccule. These primary afferents terminate in the four vestibular nuclei and in the vestibulo-cerebellum (flocculonodular lobe and vermis). Secondary sensory vestibular projections include descending upper motor neuron (UMN) axons from the medial vestibular nucleus to spinal cord lower motor neurons (LMNs) regulating head and neck movements, descending UMN axons from the lateral vestibular nucleus to LMNs at all levels to regulate extensor tone, axons from vestibular nuclei to the cerebellum, and ascending axons in the medial longitudinal fasciculus to extraocular LMNs to coordinate eye movements with head and neck movements. Some secondary sensory vestibular projections may reach the thalamus and be further conveyed to Area 2 of the postcentral gyrus for aiding in motion perception and spatial orientation. The insular cortex and temporoparietal cortex also may receive some vestibular-related projections.

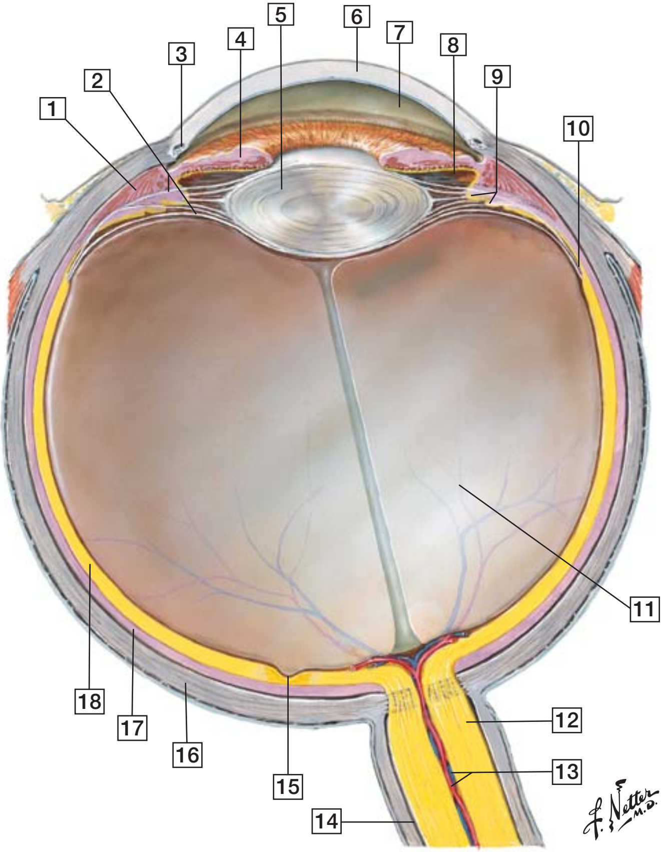

Slow phase
Rapid phase
(saccadic movement)
1
2
3
4
5
6
7
8
9
10
F. Netter M.D.

Nystagmus

1. Oculomotor neurons to the medial rectus muscle
2. Axon of abducens internuclear neuron
3. Ascending tract of Dieters into medial longitudinal fasciculus
4. Medial and lateral vestibular nuclei
5. Abducens nucleus
6. Oculomotor nerve (CN III)
7. Lateral rectus muscle
8. Medial rectus muscle
9. Parapontine reticular formation (PPRF)
10. Abducens nerve (CN VI)

Comment: Nystagmus is the repetitive, alternating, back-and-forth movement of the eyes, using circuitry to centrally coordinate extraocular lower motor neurons (LMNs). Vestibular inputs and cortical inputs regulate the control of the lateral rectus muscle on one side with the medial rectus on the other side, resulting in the coordinated movement of the eyes to maintain binocular vision. Disruption of this process results in vestibular nystagmus, in which a vestibular disruption (asymmetrical vestibular input) leaves neuronal imbalance that is interpreted as movement that requires repetitive readjustment (rapid phase, saccadic movement) to the coordinated drifting of the eyes (slow phase). In optokinetic nystagmus, a normal process, tracking movements of the eyes (e.g., to passing telephone poles while riding in a car) reaches the lateral limit of eye movement, eliciting a snap-back to the normal forward position of the eyes via signals from the association visual cortex through the superior colliculus to extraocular LMNs.

Anatomy of the Eye

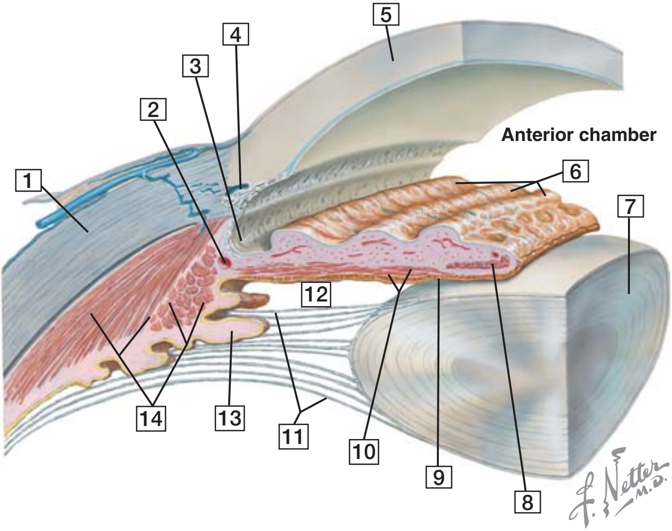


Anatomy of the Eye

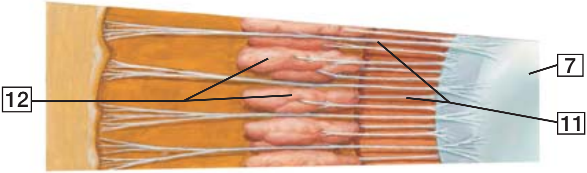
1. Ciliary body and ciliary muscle
2. Zonular fibers (suspensory ligament of the lens)
3. Scleral venous sinus (Schlemm's canal)
4. Iris
5. Lens
6. Cornea
7. Anterior chamber
8. Posterior chamber
9. Ciliary process
10. Ora serrata
11. Vitreous body
12. Optic nerve (CN II)
13. Central retinal artery and vein
14. Subarachnoid space around CN II
15. Fovea centralis of the macula
16. Sclera
17. Choroid
18. Optic portion of the retina

Comment: The eye consists of three layers or tunics: (1) outer fibrous tunic, including the cornea (transparent, responsible for 90% of light refraction in vision) and sclera (opaque); (2) middle vascular tunic (uveal tract), including the choroid, the ciliary body (from which the lens is suspended by the zonular fibers attached to the ciliary process), and the iris; and (3) the inner tunic, including the neuroretina with its photoreceptors and other neurons (by which incoming light is transduced). Aqueous humor is secreted from blood vessels of the iris into the posterior chamber and flows through the aperture of the pupil into the anterior chamber, where it is absorbed into the trabecular meshwork into Schlemm's canal at the iridocorneal angle. Blockage of this absorption results in glaucoma. The aperture of the pupil is controlled by both sympathetic (dilation, via superior cervical ganglion) and parasympathetic (constriction, via CN III from the nucleus of Edinger-Westphal) postganglionic fibers.

Anterior and Posterior Chambers of the Eye



Note: For clarity, only a single plane of zonular fibers is shown; actually, fibers surround entire circumference of the lens.



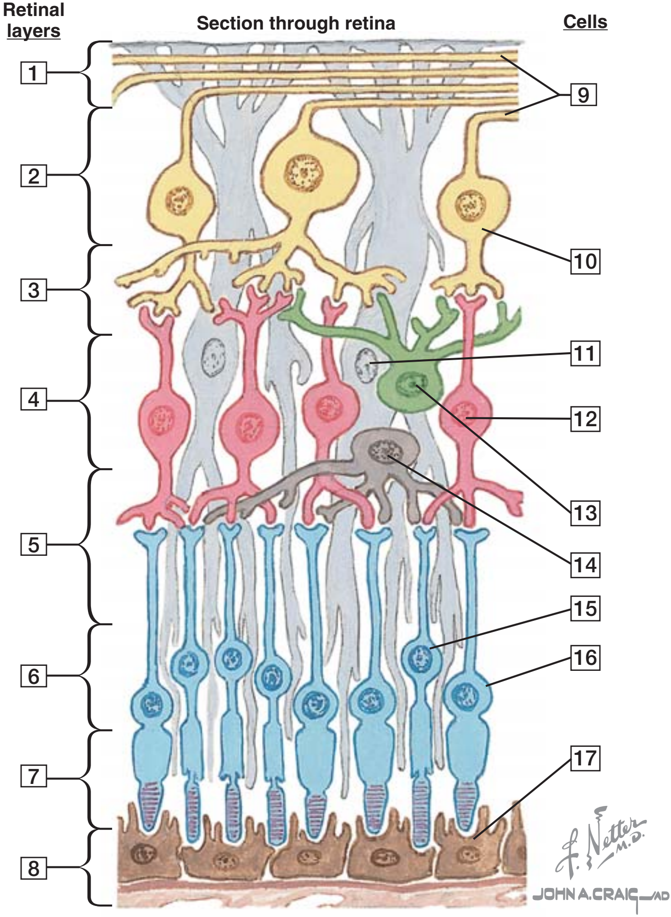
The lens and supporting structures

Anterior and Posterior Chambers of the Eye

1. Sclera
2. Major arterial circle of iris
3. Iridocorneal angle
4. Scleral venous sinus (Schlemm’s canal)
5. Cornea
6. Folds of the iris
7. Lens
8. Sphincter (constrictor) muscle of the pupil
9. Pigment epithelium of the iris
10. Dilator muscle of the pupil
11. Zonular fibers
12. Posterior chamber
13. Ciliary process
14. Ciliary muscle

Comment: The ciliary muscle is supplied by postganglionic parasympathetic fibers from the nucleus of Edinger-Westphal via CN III via the ciliary ganglion. Contraction of the ciliary muscle reduces tension on the zonular fibers of the suspensory ligament of the lens, causing the lens to curve or bunch, inducing accommodation for near vision. The pupillary constrictor muscle also is supplied by postganglionic parasympathetic fibers from CN III via the ciliary ganglion. Light shone into either eye (via CN II) activates a retinal projection to the pretectum, which sends bilateral connections to the nucleus of Edinger-Westphal via the posterior commissure. This nucleus activates the ciliary ganglion, eliciting the direct (ipsilateral) and consensual (contralateral) component of the pupillary light reflex. Compression of the third nerve may disrupt this constriction process, leading to a fixed, dilated pupil due to the unopposed action of the superior cervical ganglion on the pupillary dilator muscle.

Retina: Retinal Layers

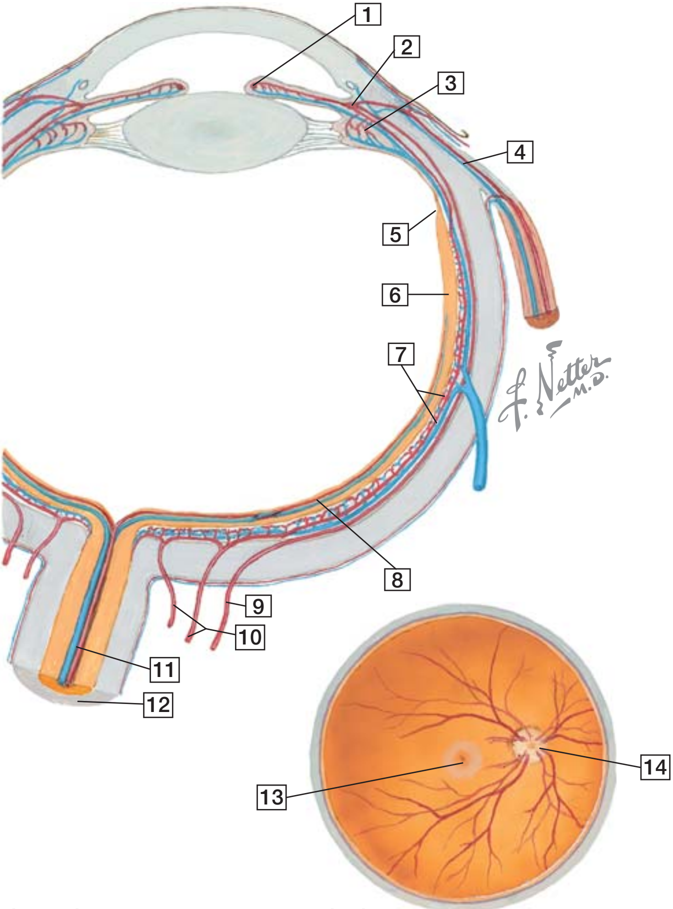


Retina: Retinal Layers

1. Nerve fiber layer
2. Ganglion cell layer
3. Inner plexiform layer
4. Inner nuclear layer
5. Outer plexiform layer
6. Outer nuclear layer
7. Photoreceptor layer
8. Pigment epithelium
9. Axonal layer (inner) of the retina
10. Ganglion cell
11. Muller cell (supporting glial cell)
12. Bipolar cell
13. Amacrine cell
14. Horizontal cell
15. Rod
16. Cone
17. Pigment cells of the choroids

Comment: The retina is a tissue-paper thin piece of central nervous system tissue that contains the photoreceptors that transduce incoming light energy to electrical messages send into the brain. It is attached to the vascular tunic at the ora serrata. The center of the macula (dense photoreceptors) consists entirely of cones; the cones connect with little convergence on bipolar neurons, which in turn synapse on retinal ganglion cells. These ganglion cells project to the pretectum, superior colliculus, lateral geniculate nucleus, suprachiasmatic nucleus of the hypothalamus, and other structures. This macular pathway (through the lateral geniculate nucleus) is responsible for color vision (photopic, high acuity). Rods are the main photoreceptor of the retina and have extensive convergence onto bipolar neurons, with further connections to ganglion cells. The rods are responsible for night vision (scotopic, lower acuity). The macula can undergo a gradual process of depigmentation and degeneration with age (macular degeneration), leading to the loss of central vision and inability to read.

Arteries and Veins of the Eye



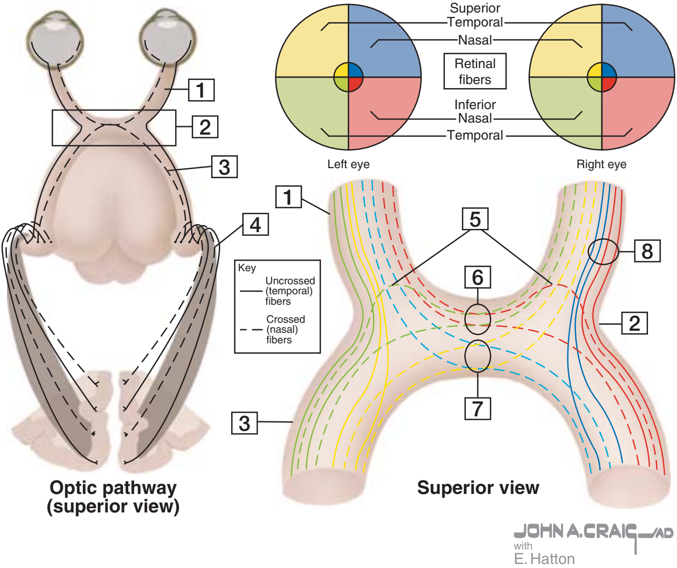
Right retinal vessels: ophthalmoscopic view

Arteries and Veins of the Eye

1. Minor arterial circle of the iris
2. Major arterial circle of the iris
3. Blood vessels of the ciliary body
4. Anterior ciliary artery and vein
5. Ora serrata
6. Retina
7. Choroid
8. Retinal artery and vein
9. Long posterior ciliary artery
10. Short posterior ciliary arteries
11. Central retinal artery and vein
12. Optic nerve (CN II)
13. Macula and fovea centralis
14. Optic disc

Comment: The central retinal artery, derived from the ophthalmic artery (first branch of the internal carotid artery), supplies blood to the retina; it is the site where ischemic or embolic events (transient ischemic attacks [TIAs]) herald the presence of serious vascular disease and high risk for future stroke. Ciliary arteries supply the middle vascular tunic; this component of the retinal blood supply can be disrupted by a detached retina. The blood vessels enter and exit the retina at the optic disc (nerve head), located nasally and slightly inferiorly from the geometric midpoint of the eyeball.

See book 14.26



Optic pathway (superior view)

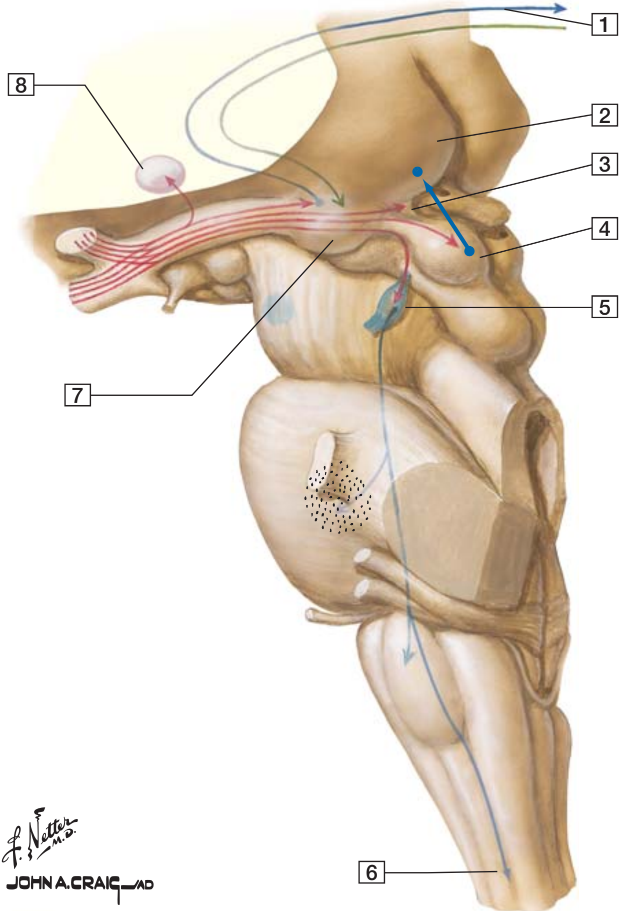
Superior view

Optic Chiasm

1. Optic nerve
2. Optic chiasm
3. Optic tract
4. Optic radiations
5. Inferior nasal fibers after decussation
6. Inferior nasal fibers of the optic chiasm
7. Superior nasal fibers of the optic chiasm
8. Temporal fibers remaining ipsilateral

Comment: Axons from ganglion cells in the temporal hemiretinas travel in the optic nerve and remain ipsilateral through the optic chiasm into the optic tract. Axons from ganglion cells in the nasal hemiretinas travel in the optic nerve and decussate through the optic chiasm to enter the contralateral optic tract. Therefore, information from the nasal hemiretina of one eye is coordinated with information from the temporal hemiretina of the other eye. These ganglion cell projections terminate in the lateral geniculate nucleus, which further processes the information and projects to the primary visual cortex in Area 17 of the occipital lobe. Binocular convergence occurs in this cortical area. Damage to the optic chiasm results in disruption of crossing axons from the nasal hemiretinas. This produces bitemporal hemianopia, starting first as an upper visual quadrant defect and then progressing to full hemianopia. The optic tract contains axons from the ipsilateral temporal hemiretina and contralateral nasal hemiretina, representing the contralateral visual field (visual world). Disruption of the optic tract results in contralateral hemianopia.

Visual Pathways: Retinal Projections to the Thalamus, Hypothalamus, and Brain Stem



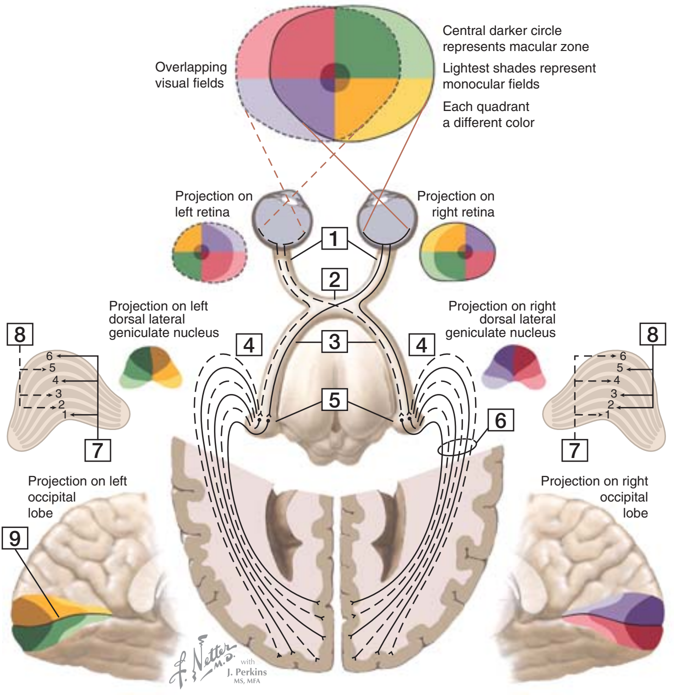
Visual Pathways: Retinal Projections to the Thalamus, Hypothalamus, and Brain Stem

1. Axonal projections to visual cortex
2. Pulvinar
3. Pretectum
4. Superior colliculus
5. Nucleus of the accessory optic tract
6. Projections to preganglionic sympathetic neurons in T1 and T2
7. Lateral geniculate nucleus (body)
8. Suprachiasmatic nucleus (hypothalamus)

Comment: Ganglion cells of the retina send axons (equivalent to lemniscal pathways of other sensory systems) through the optic nerve, chiasm, and tract, to terminate in the lateral geniculate nucleus (LGN) of the thalamus, superior colliculus, pretectum, suprachiasmatic nucleus of the hypothalamus, and some brain stem nuclei such as the nucleus of the accessory optic tract. The pathway through the LGN mediates conscious interpretation of the visual world. The pathway through the superior colliculus provides additional localizing information regarding vision and mediates reflex visual effects on head and neck movement. The pathway through the pretectum mediates the pupillary light reflex. The pathway through the suprachiasmatic nucleus integrates total light flux and regulates circadian rhythms and diurnal cycles. The pathway through the nucleus of the inferior accessory optic tract may help to mediate brain stem responses for visual tracking and helps to mediate sympathetic preganglionic neurons in the T1 and T2 spinal cord.

See book 14.28

Visual Pathway: Retino-Geniculo-Calcarine Pathway


Overlapping visual fields
Central darker circle represents macular zone
Lightest shades represent monocular fields
Each quadrant a different color
Projection on left retina
Projection on right retina
1
2
Projection on left dorsal lateral geniculate nucleus
Projection on right dorsal lateral geniculate nucleus
8
6
5
4
3
2
1
7
4
3
4
5
6
8
6
5
4
3
2
1
7
Projection on left occipital lobe
9
Projection on right occipital lobe
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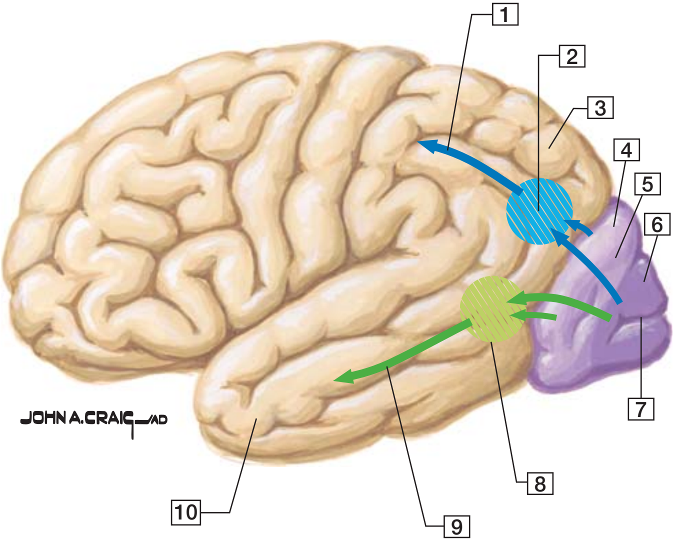
Visual Pathway: Retino-Geniculo-Calcarine Pathway

1. Optic nerves (CN II)
2. Optic chiasm
3. Optic tracts
4. Meyer's loop
5. Lateral geniculate nuclei (bodies)
6. Optic radiations
7. Contralateral input to the lateral geniculate nucleus (LGN) from contralateral nasal hemiretina
8. Ipsilateral input to the LGN from ipsilateral temporal hemiretina
9. Calcarine fissure

Comment: The retino-geniculo-calcarine pathway conveys information for fine-grained visual analysis of the outside world. It is organized topographically (retinotopic) throughout the entire pathway. Through the crossing axons (optic chiasm) of the ganglion cells in the nasal hemiretinas and the ipsilateral axons of the ganglion cells in the temporal hemiretinas, the left visual field is represented in the right LGN and visual cortex, and the right visual field is represented in the left LGN and visual cortex. Ipsilateral and contralateral ganglion cell axons remain segregated in the LGN; binocular convergence takes place in Area 17 of the primary visual cortex. The optic radiations, in their course through the cortical white matter, loop into the temporal lobe, especially axons representing the contralateral upper quadrant of vision. Damage to these axons of Meyer's loop in the temporal lobe results in a contralateral upper quadrantanopia.

Visual Pathways in the Parietal and Temporal Lobes

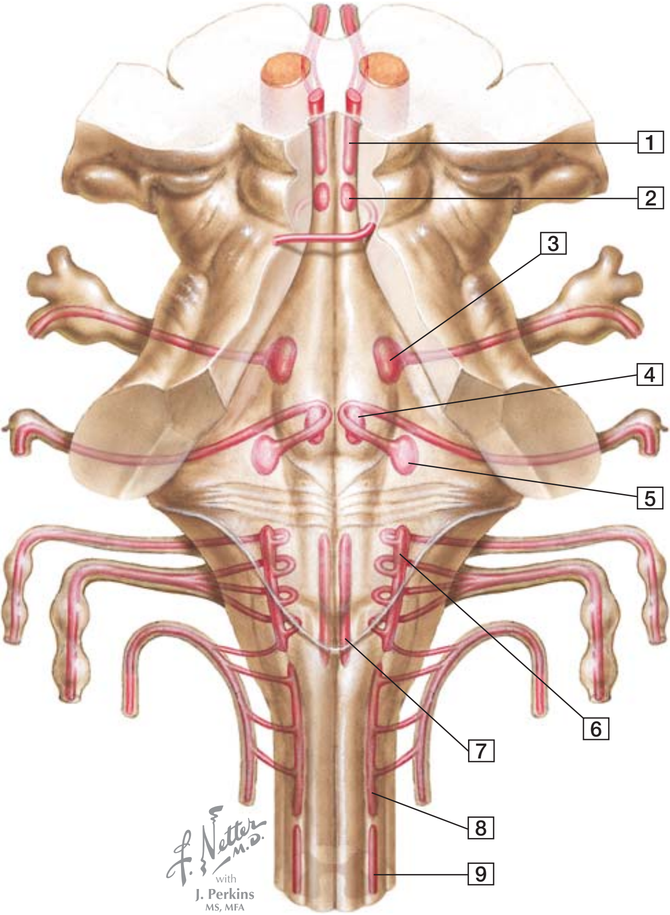


Visual Pathways in the Parietal and Temporal Lobes

1. Spatial visual pathway
2. Middle temporal area
3. Parietal lobe
4. Association visual cortex V3
5. Association visual cortex V2
6. Occipital lobe
7. Primary visual cortex V1
8. V4 visual cortex
9. Object recognition pathway
10. Temporal lobe

Comment: Neurons in the primary visual cortex (V1, Area 17) send axons to association visual cortices (V2 and V3, Areas 18 and 19); Areas 18 and 19 also receive inputs from the superior colliculus via the pulvinar. V1, V2, and V3 project to the middle temporal area (MTA) and V4. The MTA neurons are direction selective and motion responsive and further project into the parietal lobe for spatial visual processing related to motion and positional relationships among objects in the visual field. V4 neurons are involved in shape and color perception and project to further temporal lobe structures to provide high-resolution object recognition, including faces, animate and inanimate objects, and the classification and orientation of objects. Small vascular infarcts in the temporal lobe may produce specific agnosias such as an inability to recognize specific types of objects (faces, animate objects) or their orientation.

Distribution of Lower Motor Neurons (LMNs) in the Brain Stem

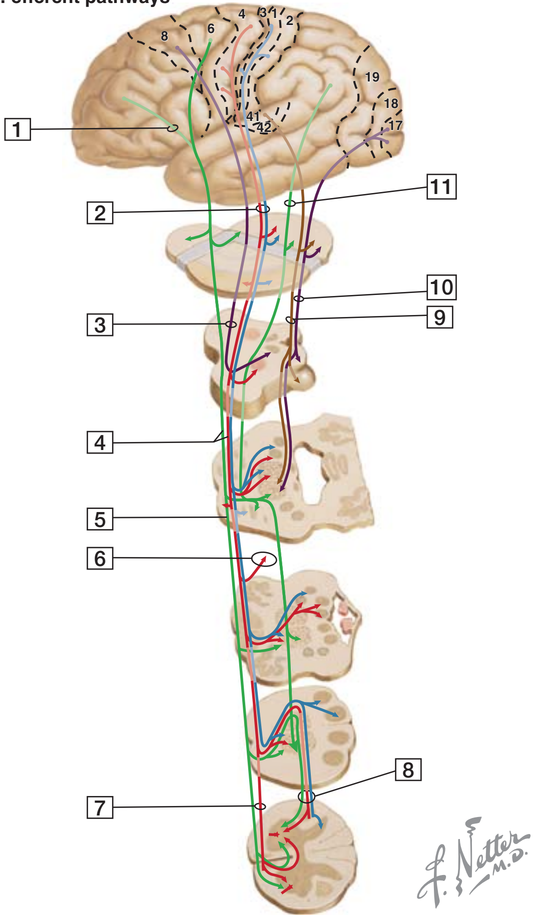


Distribution of Lower Motor Neurons (LMNs) in the Brain Stem

1. Oculomotor nucleus
2. Trochlear nucleus
3. Trigeminal motor nucleus
4. Abducens nucleus
5. Facial nucleus
6. Nucleus ambiguus
7. Hypoglossal nucleus
8. Spinal accessory nucleus
9. Spinal cord ventral horn LMNs

Comment: Lower motor neurons (LMNs) are found in a medial column and a lateral column in a longitudinal view of the brain stem. The medial column consists of the extraocular nuclei (oculomotor, trochlear, abducens nuclei) and the hypoglossal nucleus, all derived from the general somatic efferent system. The lateral column consists of the trigeminal motor nucleus, facial nucleus, nucleus ambiguus, and spinal accessory nucleus, all derived from the special visceral efferent system. All of these nuclei contain classical LMNs with axonal connections leaving the CNS and terminating with neuromuscular junctions on skeletal muscle fibers. The LMNs of the spinal cord are found at every level in clusters in the ventral horn, with the proximal extremities and trunk represented more medially and the distal musculature represented more laterally and dorsally.

Cerebral cortex: efferent pathways

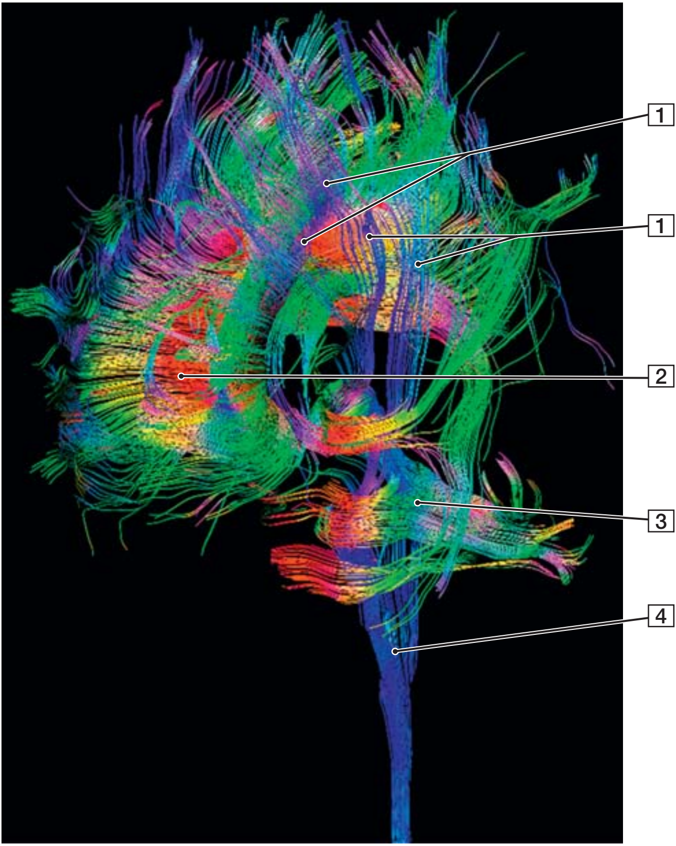


Cortical Efferent Pathways

1. Axons from frontal cortex
2. Corticobulbar, corticorubral, corticonuclear, and corticospinal pathways
3. From frontal eye fields to interstitial nucleus of Cajal
4. Corticospinal axons
5. Cortical axons to pontocerebellar system
6. Axons to contralateral facial nerve nucleus
7. Anterior corticospinal tract (uncrossed)
8. Lateral corticospinal tract (crossed)
9. Axons from auditory cortex to inferior colliculus
10. Axons from occipital eye fields to superior colliculus
11. Axons from parietal cortex

Comment: Cortical neurons of the motor, premotor, and supplemental motor cortex send axons to the basal ganglia (caudate nucleus and putamen), nuclei ventral anterior (VA) and ventolateral (VL) of the thalamus, the red nucleus, the pontine nuclei, cranial nerve motor nuclei of both sides (corticobulbar), and the spinal cord ventral horn (mainly contralateral). These axons form the corticospinal tracts, corticobulbar tract, corticostriatal projections, corticopontine projections, corticothalamic projections, and cortical connections to brain stem upper motor neuronal systems. Cortical neurons of the sensory cortex connect mainly to secondary sensory nuclei to regulate incoming sensory information (corticonuclear path). Neurons in the frontal eye fields project to the superior colliculus, the horizontal and vertical gaze centers of the brain stem, and the interstitial nucleus of Cajal in the midbrain to coordinate voluntary eye movements and associated head movements. Other cortical projections connect with the thalamus, brain stem structures, and limbic forebrain regions.

Lateral / oblique view

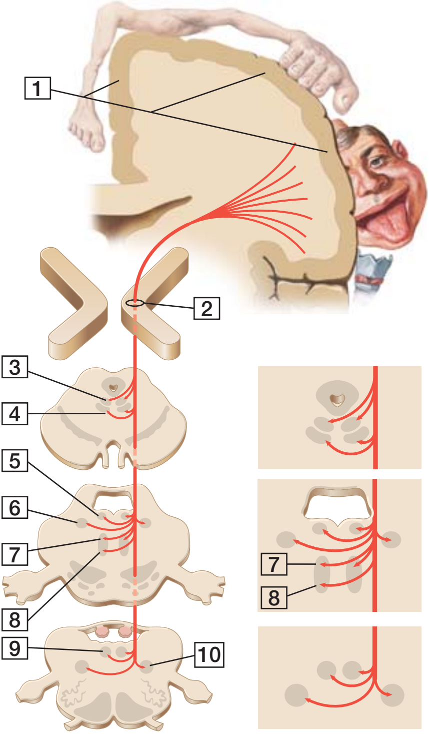


Color Imaging of Cortical Efferent Pathways

1. Cortical efferent fibers
2. Midline fibers of the corpus callosum
3. Superior cerebellar peduncle
4. Pyramids

Comment: Diffusion tensor imaging demonstrates cortical efferent pathways, shown in blue. Cortical efferent fibers from widespread regions of the cerebral cortex project to structures in the forebrain (e.g., basal ganglia), thalamus, brain stem, cerebellum, and spinal cord. Additional cortical association pathways are depicted in green, and commissural pathways are depicted in red.

Corticobulbar Tract



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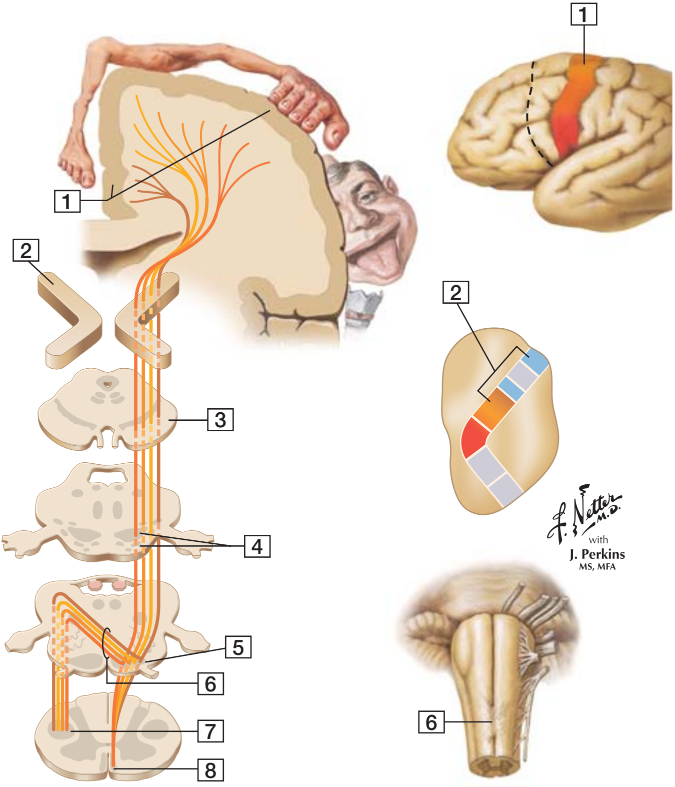
Corticobulbar Tract

1. Primary motor cortex (Area 4)
2. Genu of the internal capsule
3. Oculomotor nucleus
4. Trochlear nucleus
5. Abducens nucleus
6. Trigeminal motor nucleus
7. Facial nucleus to the upper face
8. Facial nucleus to the lower face
9. Hypoglossal nucleus
10. Nucleus ambiguus

Comment: The corticobulbar tract arises mainly from the lateral portion of the primary motor cortex (Area 4). Corticobulbar axons project through the genu of the internal capsule, cerebral peduncle, basis pontis, and medullary pyramids on the ipsilateral side. Corticobulbar axons terminate in the cranial nerve motor nuclei on both sides except for the portion of the facial nucleus that distributes to muscles of the lower face; that nucleus receives exclusively contralateral projections. Therefore, a corticobulbar tract lesion (such as occurs with a stroke affecting the genu of the internal capsule, or a cerebral peduncle lesion) results in a contralateral drooping lower face whose muscles cannot contract on command (central facial palsy). This can be distinguished from a peripheral facial palsy (Bell's palsy), which demonstrates total facial paralysis of both upper and lower musculature. In some patients the corticobulbar projections to the hypoglossal nucleus are mainly contralateral, and to the spinal accessory nucleus are mainly ipsilateral; however, these anatomical observations usually are not accompanied by long-term functional consequences.

Corticospinal Tract



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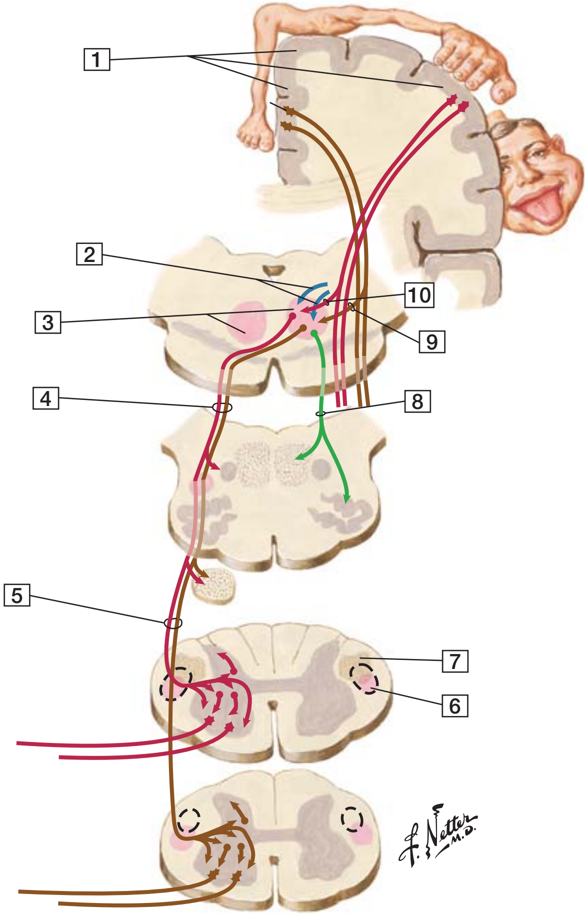
Corticospinal Tract

1. Primary motor cortex (Area 4)
2. Posterior limb of the internal capsule
3. Cerebral peduncle
4. Basis pontis
5. Medullary pyramid
6. Decussation of the pyramids
7. Lateral corticospinal tract (crossed)
8. Anterior corticospinal tract (uncrossed)

Comment: The motor portion of the corticospinal (CST) tract arises mainly from the primary motor cortex (Area 4) and the supplemental and premotor cortices (Area 6). The CST descends in the posterior limb of the internal capsule, cerebral peduncle, basis pontis, and medullary pyramids. Approximately 80% of the CST decussates in the pyramidal decussation at the junction of the medulla and the spinal cord, continues as the lateral CST, and terminates on lower motor neurons (LMNs) in the contralateral ventral horn of the spinal cord. The remaining 20% remains uncrossed in the anterior CST and then mainly crosses in the anterior white commissure at the level of termination in the spinal cord to innervate LMNs in the medial portion of the ventral horn. Most of the CST terminations end on interneurons associated with LMNs, although approximately 10% of the terminations are monosynaptic on LMNs. The CST system is directed to both alpha and gamma LMNs, thereby assuring that skeletal muscle contractions brought about by this upper motor neuron (UMN) influence are accompanied by concomitant adjustments of the muscle spindles to keep them responsive. Damage to the CST system is usually accompanied by additional involvement of corticorubral (and other cortical efferent) fibers (involvement in the internal capsule) or rubrospinal tract fibers (lateral funiculus of the spinal cord); such a lesion results in contralateral spastic hemiplegia with hypertonus, hyperreflexia, and pathological reflexes (plantar extensor responses, clonus). In the rare instances of pure CST lesions in the medullary pyramids, the resultant contralateral deficit shows clumsiness of hand and finger movements with mild loss of tone, not the classical UMN spastic hemiplegia.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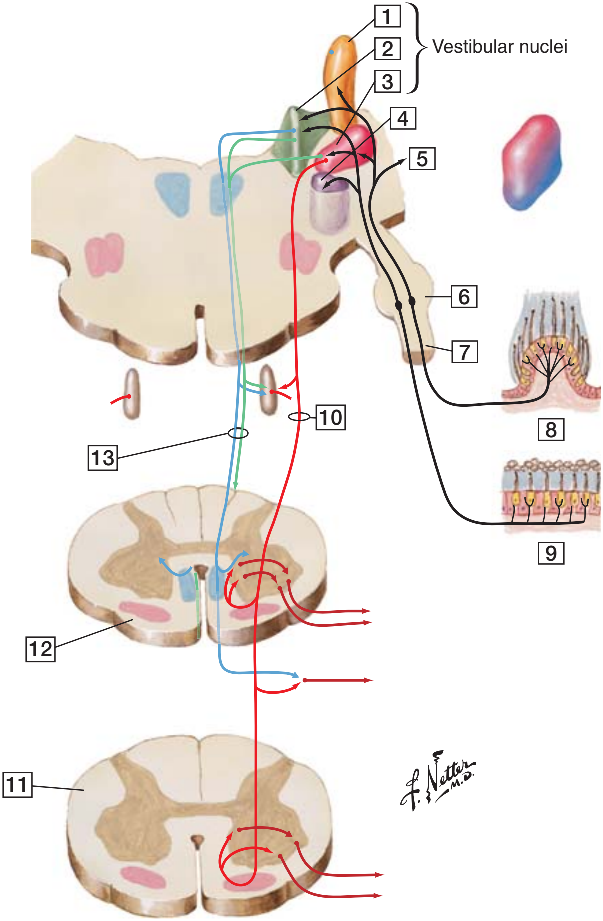
Rubrospinal Tract

1. Primary motor cortex (Area 4)
2. Fibers from deep cerebellar nuclei (globose, emboliform, some from dentate)
3. Red nucleus
4. Crossed rubrospinal and rubromedullary fibers
5. Rubrospinal tract
6. Rubrospinal tract
7. Lateral corticospinal tract
8. Uncrossed rubromedullary fibers
9. Cortical influences helping to drive flexor actions of the rubrospinal system for lower extremities
10. Cortical influences holding in check flexor actions of the rubrospinal system on upper extremities

Comment: The cortico-rubro-spinal system is an indirect corticospinal (CST) system influencing movements of the extremities. The red nucleus in the midbrain receives topographically organized ipsilateral connections from the primary motor cortex (Area 4). Axons of the rubrospinal tract (RST) decussate in the ventral tegmental decussation in the midbrain and descend in the lateral brain stem and the lateral funiculus of the spinal cord, where they overlap extensively with the lateral CST. The RST terminates mainly on interneurons associated with alpha and gamma lower motor neurons (LMNs) in the spinal cord, especially those associated with flexor movements. Through cortical influences on the RST, this tract helps to drive flexor movements of the upper extremity and hold in check flexor movements of the lower extremity. RST lesions usually occur in conjunction with damage to the corticorubral axons or the CST; these lesions contribute to the upper motor neuron syndrome of contralateral spastic hemiplegia with hypertonus, hyperreflexia, and pathological reflexes. Brain stem lesions caudal to the red nucleus result in decerebration (extensor spasticity of all four limbs), reflecting the removal of the flexor drive of the RST on LMNs supplying the upper limb.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Vestibular nuclei
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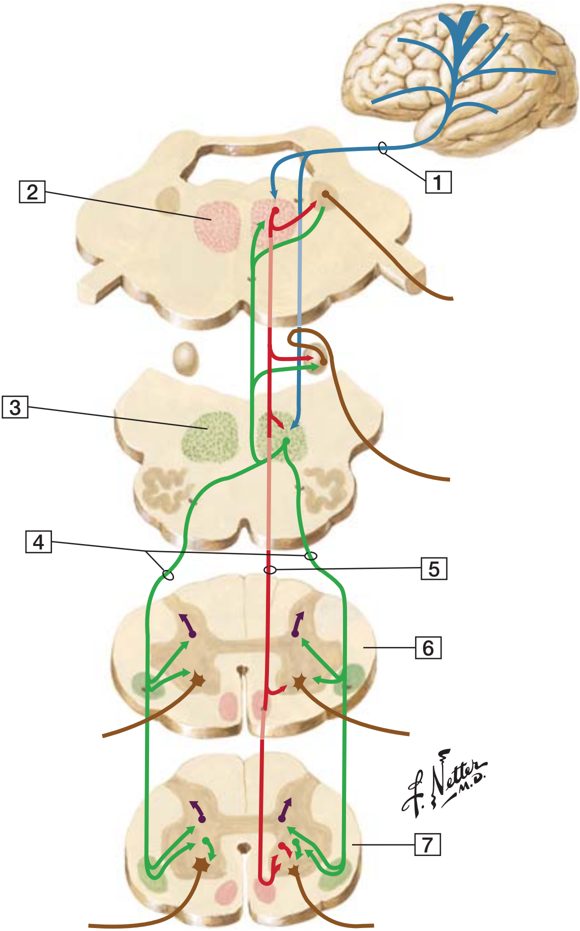
Vestibulospinal Tracts

1. Superior vestibular nucleus
2. Medial vestibular nucleus
3. Lateral vestibular nucleus
4. Inferior vestibular nucleus
5. Primary vestibular projections to the cerebellum
6. Vestibular ganglion
7. Vestibular nerve (CN VIII)
8. Fibers from the cristae ampullaris
9. Fibers from the maculae
10. Lateral vestibulospinal tract
11. Lumbar part of the spinal cord
12. Lower part of the cervical spinal cord
13. Medial vestibulospinal tract

Comment: The lateral vestibulospinal tract arises from the lateral vestibular nucleus and terminates mainly on interneurons associated with ipsilateral alpha and gamma lower motor neurons (LMNs) associated with extensor (antigravity) musculature, especially proximal musculature. This powerful antigravity system, driven by primary vestibular input, is held in check by the red nucleus and by connections from the cerebellum. Removal of these influences by damage to the anterior cerebellum (alcoholic damage) or brain stem damage caudal to the red nucleus but rostral to the lateral vestibular nucleus results in a constant state of extensor hypertonus, especially with the latter type of lesion. Such brain stem damage is referred to as *decerebrate posturing*, with all four limbs extended and the upper extremities internally rotated. The medial vestibulospinal tract arises from the medial vestibular nucleus and provides inhibition of alpha and gamma LMNs (via interneurons) controlling neck and axial musculature. These two vestibulospinal tracts stabilize and coordinate the position of the head, neck, and body and provide important reflex and brain stem control over tone and posture. These vestibulospinal systems function in coordinated fashion with the reticulospinal systems.

See book 15.10

Reticulospinal Tracts



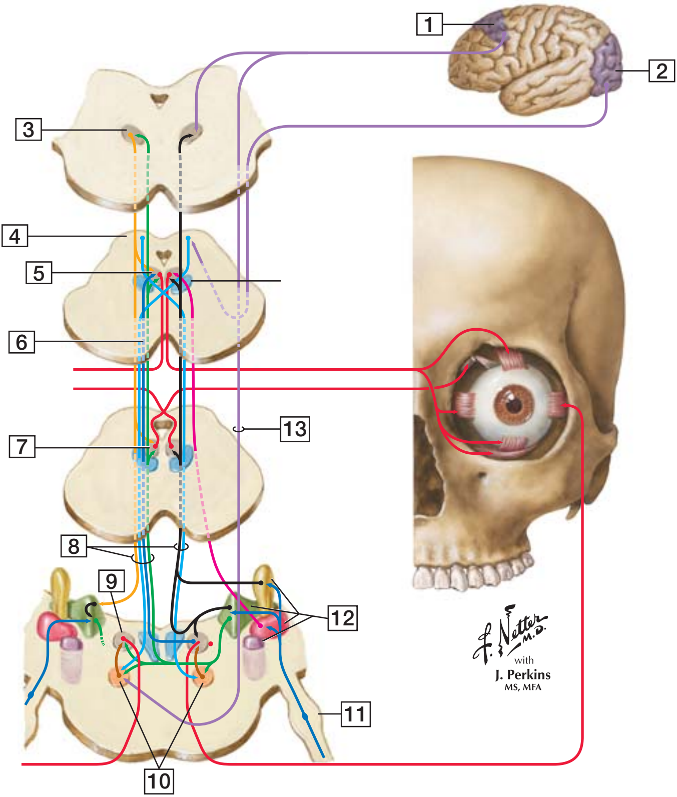
Reticulospinal Tracts

1. Corticoreticular projections
2. Medial pontine reticular formation
3. Medial medullary reticular formation
4. Lateral reticulospinal tract
5. Medial reticulospinal tract
6. Cervical spinal cord
7. Lumbar spinal cord

Comment: The reticulospinal tracts are derived from the isodendritic neurons in the medial portion of the pontine and medullary reticular formation. The pontine (medial) reticulospinal tract arises from the medial pontine reticular formation (nuclei pontis caudalis and pontis oralis) and descends ipsilaterally to end mainly on interneurons associated with alpha and gamma lower motor neurons (LMNs) of the spinal cord at all levels; this tract has an extensor bias for axial musculature and reinforces the actions of the lateral vestibulospinal tract. This tract is driven mainly by polysensory input from the trigeminal and somatosensory systems and has little influence from the cerebral cortex. The medullary (lateral) reticulospinal tract arises from the medial reticular formation of the medulla (nucleus gigantocellularis) and is driven heavily by cortical input from the motor cortex, supplemental motor cortex, and premotor cortex. Axons of this tract terminate bilaterally mainly on interneurons associated with alpha and gamma LMNs at all levels of the spinal cord; this tract exerts a flexor bias, reinforcing the corticospinal and rubrospinal tracts. These reticulospinal tracts are important regulators of basic tone and posture. They appear to not be somatotopically organized.

See book 15.11

Central Control of Eye Movements

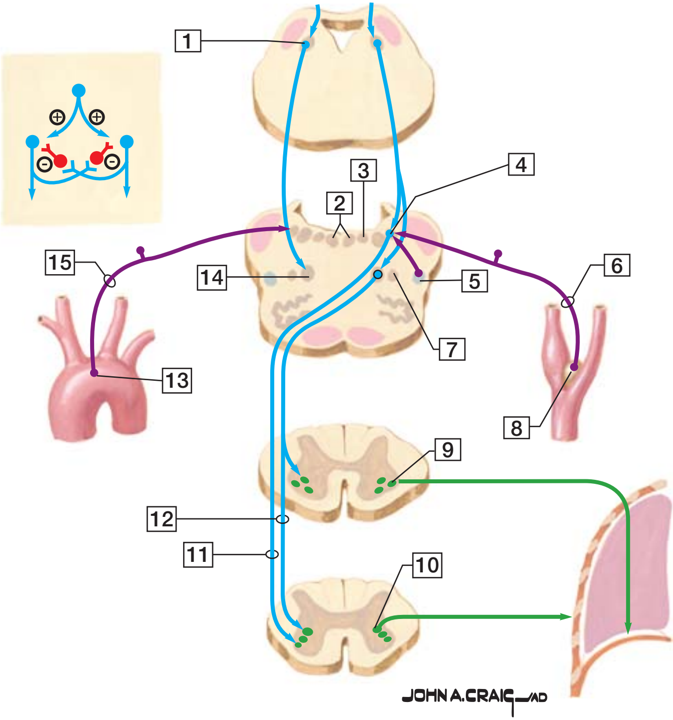


Central Control of Eye Movements

1. Frontal eye fields (Area 8)
2. Occipital eye fields (Areas 17, 18, 19)
3. Interstitial nucleus of Cajal
4. Superior colliculus
5. Oculomotor nucleus
6. Abducens internuclear neuronal projection
7. Trochlear nucleus
8. Medial longitudinal fasciculi
9. Abducens nucleus
10. Parapontine reticular formation (PPRF)
11. Vestibular ganglion and nerve
12. Vestibular nuclei
13. Corticoreticular fibers

Comment: Central control of eye movements is achieved through the coordination of extraocular motor nuclei for CNs III, IV, and VI; this is achieved through the parapontine reticular formation (PPRF, horizontal gaze center). The PPRF receives input from the vestibular nuclei, the deep layers of the superior colliculus (reflecting inputs from V1, V2, and V3), the frontal eye fields of the cerebral cortex, and the interstitial nucleus of Cajal (reflecting inputs from the vestibular nuclei and the frontal eye fields). The PPRF drives the ipsilateral abducens nucleus for movement of the lateral rectus muscle, and the contralateral oculomotor nucleus (via interneurons in the abducens nucleus) for movement of the medial rectus muscle of the contralateral side, thereby coordinating horizontal eye movements. The interstitial nucleus of Cajal helps to coordinate vertical and oblique eye movements. Secondary sensory vestibular projections also terminate in the extraocular motor CNN. Axons interconnecting the extraocular motor nuclei travel through the medial longitudinal fasciculus.

Central Control of Respiration

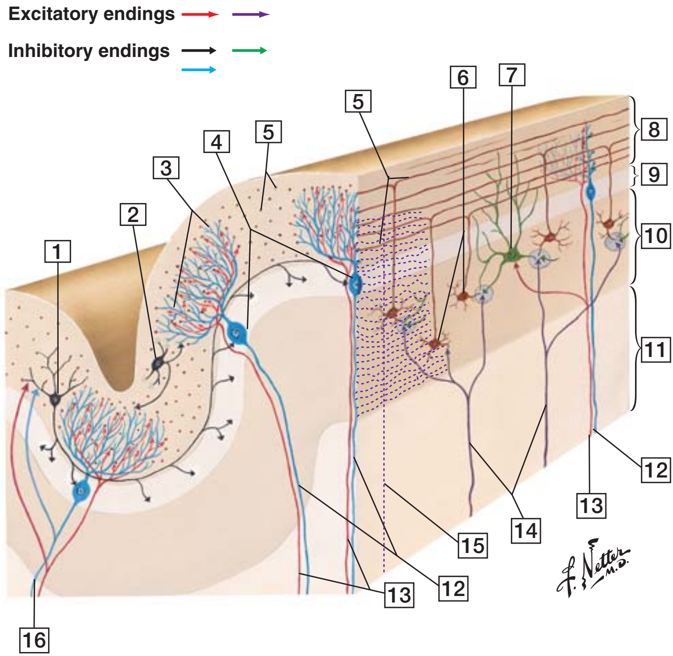


Central Control of Respiration

1. Medial parabrachial nucleus
2. Hypoglossal nucleus
3. Dorsal motor nucleus of CN X
4. Dorsal respiratory nucleus (ventrolateral nucleus solitarius)
5. Central chemoreceptor zone
6. CN IX
7. Nucleus ambiguus
8. Carotid body
9. Lower motor neurons (LMNs) of phrenic nucleus
10. LMNs for intercostals and accessory muscles of respiration
11. Polysynaptic connections to LMNs for inspiratory muscles
12. Polysynaptic connections to LMNs for expiratory muscles
13. Aortic body
14. Ventral respiratory nucleus (nucleus retroambiguus)
15. CN X

Comment: Inspiration and expiration are regulated by nuclei of the reticular formation. The dorsal respiratory nucleus (the ventrolateral nucleus solitarius) sends crossed axons to terminate on cervical spinal cord lower motor neurons (LMNs) of the phrenic nucleus and on thoracic spinal cord LMNs that supply intercostal muscles and accessory musculature associated with inspiration. This nucleus receives input from the carotid body (via CN IX), from the aortic body chemoreceptors (via CN X), and from central chemoreceptive zones of the lateral medulla. The ventral respiratory nucleus (nucleus retroambiguus) sends crossed axons to terminate on thoracic LMNs that supply accessory musculature associated with expiration. The dorsal and ventral respiratory nuclei inhibit each other. The medial parabrachial nucleus acts as a respiratory pacemaker to regulate the dorsal and ventral respiratory nuclei. The medial parabrachial nucleus is itself regulated from higher centers such as the amygdala and the cerebral cortex.

Cerebellar Neuronal Circuitry

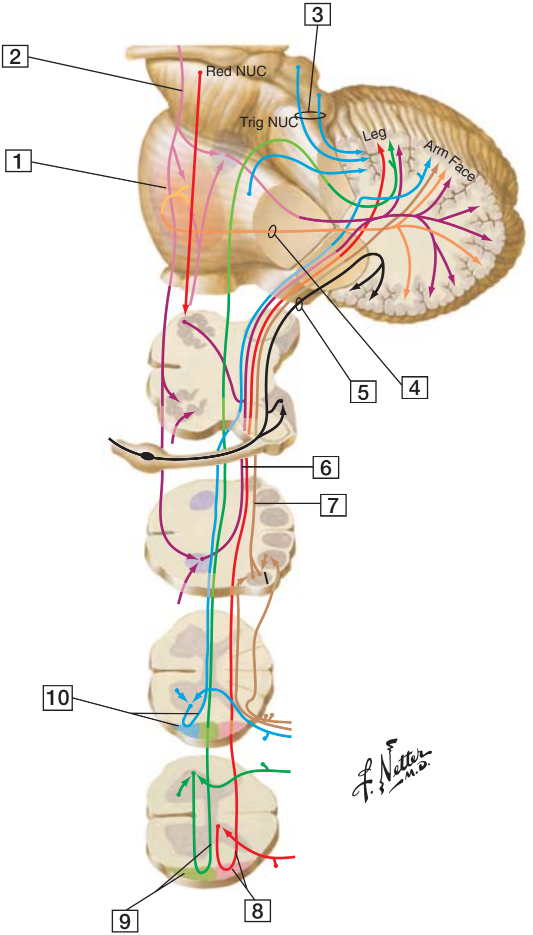


Cerebellar Neuronal Circuitry

1. Basket cell
2. Outer stellate cell
3. Dendrites of Purkinje cells
4. Purkinje cells
5. Parallel fibers
6. Granule cells
7. Golgi cells
8. Molecular layer
9. Purkinje cell layer
10. Granule layer
11. White matter
12. Purkinje cell axon
13. Climbing fiber
14. Mossy fibers
15. Locus coeruleus axon (noradrenergic)
16. Purkinje axons to deep cerebellar nuclei

Comment: The cerebellum is organized into four parts: an outer three-layered cortex, white matter, deep cerebellar nuclei, and cerebellar peduncles that connect the cerebellum with the spinal cord, brain stem, and thalamus. Purkinje cells are the major neurons in the cerebellar cortex, receiving input from climbing fibers (from the inferior olivary nuclei) and mossy fibers (via granule cells receiving all other central nervous system inputs). The Purkinje cell dendritic trees are arranged in parallel plates; the parallel fibers from granule cells span up to several hundred Purkinje cell dendritic trees. Additional cerebellar cortical neurons (basket cells, stellate cells, Golgi cells) regulate the excitability of Purkinje cell responses. Noradrenergic inputs from the locus coeruleus modulate the excitability of neurons in all three layers. The Purkinje cell axons project to the deep cerebellar nuclei and control their outputs to upper motor neuronal systems.

Afferent Pathways to the Cerebellum

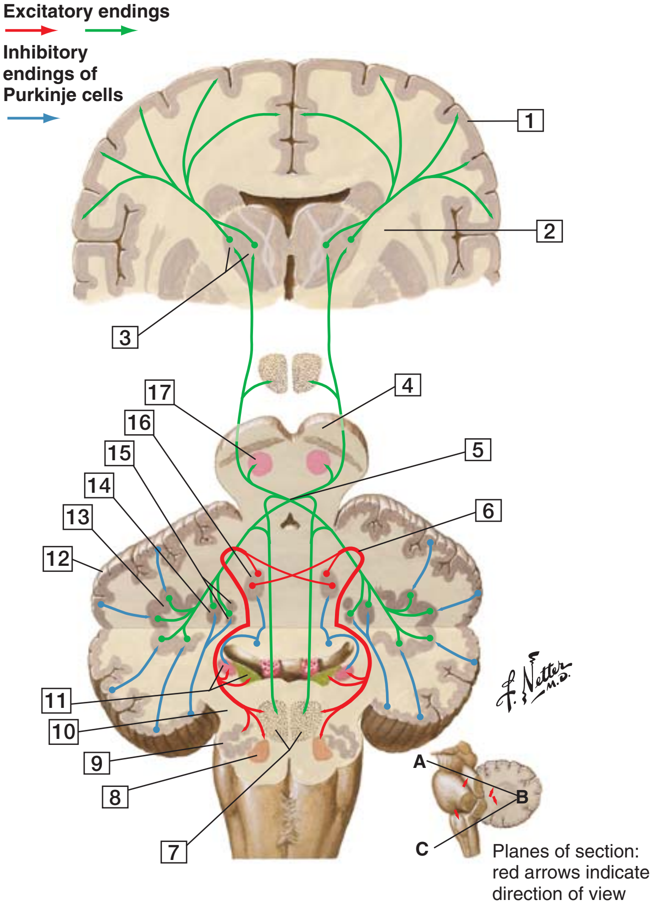


Afferent Pathways to the Cerebellum

1. Pontine nuclei
2. Cerebral cortical input
3. Tectocerebellar input
4. Pontocerebellar input
5. Vestibulospinal input
6. Reticulocerebellar tract
7. Cuneocerebellar tract
8. Dorsal spinocerebellar tract
9. Ventral spinocerebellar tract
10. Rostral spinocerebellar tract

Comment: Afferents to the cerebellum terminate in both the deep nuclei (aiding coarse adjustment) and the cerebellar cortex (aiding fine adjustment) in topographically organized zones (at least three body representations in the cerebellar cortex). Afferents travel through the cerebellar peduncles. The inferior cerebellar peduncle conveys spinocerebellar pathways (dorsal, rostral, cuneocerebellar); inferior olivary input; reticular formation input; primary and secondary vestibular input; and some trigeminal input. These inputs are mainly ipsilateral. The middle cerebellar peduncle conveys mainly crossed pontocerebellar input. The superior cerebellar peduncle conveys the ventral spinocerebellar tract (crosses twice), visual and auditory tectocerebellar input, some trigeminal input, and noradrenergic input from the locus coeruleus. These afferent inputs continuously inform the cerebellum about the state of the periphery (muscles, tendons, joints, other sensory inputs, reticular formation state), as well as the ongoing motor plans (especially from the cerebral cortex via the pontine nuclei), to permit a comparator function to allow smoothing and coordinating of motor activities via cerebellar outputs from the deep nuclei to the upper motor neuronal systems. The cerebellum also is involved in coordinating neuronal activities of nonmotor systems of the central nervous system.

Cerebellar Efferent Pathways



Planes of section: red arrows indicate direction of view

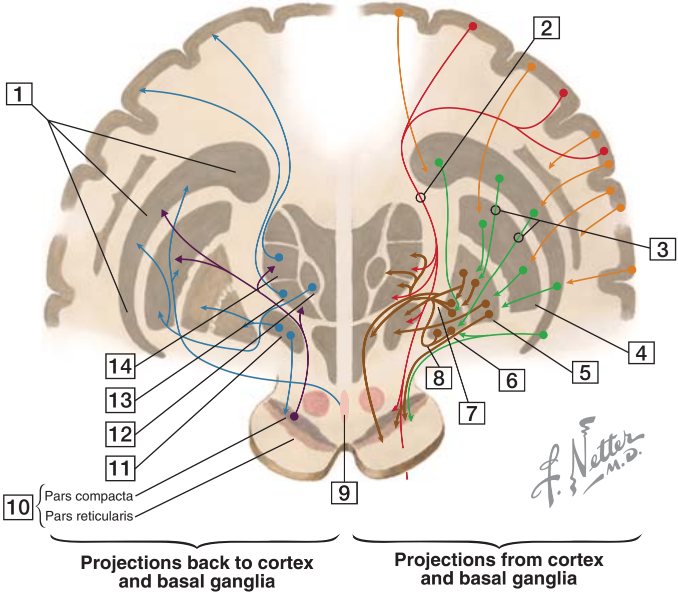
Cerebellar Efferent Pathways

1. Motor and premotor cerebral cortex
2. Internal capsule
3. Ventral anterior and ventral lateral thalamic nuclei
4. Cerebral peduncle
5. Decussation of superior cerebellar peduncles (SCPs)
6. Hook bundle of Russell
7. Pontomedullary reticular formation
8. Lateral reticular nucleus
9. Inferior olive
10. Inferior cerebellar peduncle (ICP)
11. Vestibular nuclei
12. Cerebellar cortex
13. Dentate nucleus
14. Emboliform nucleus
15. Globose nucleus
16. Fastigial nucleus
17. Red nucleus

Comment: Efferents from the cerebellum derive from the deep nuclei. Projections from the fastigial nuclei exit mainly through the ICP and terminate ipsilaterally in the lateral vestibular nucleus and pontine and medullary reticular nuclei, modulating activity of the vestibulospinal and reticulospinal upper motor neuron (UMN) systems. Axons from the globose and emboliform nuclei exit through the SCP and terminate mainly contralaterally in the red nucleus and to a lesser extent in nucleus ventral lateral (VL) of the thalamus, modulating activity of the rubrospinal UMN system. Axons from the dentate nucleus exit through the SCP and terminate contralaterally (via the decussation of the SCP) mainly in the VL nucleus of the thalamus, and to a lesser extent in the ventral anterior (VA) nucleus, modulating activity of the corticospinal tract through the thalamic (VL, VA) regulation of the motor, premotor, and supplemental motor cortices. These connections, in conjunction with the projections of the UMN system that they modulate, result in the cerebellum exerting ipsilateral smoothing and coordination of movement related to motor activities. The cerebellum also is involved with sensory, autonomic, and other functional activities of the central nervous system.

Connections of the Basal Ganglia

Connections of the basal ganglia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Pars compacta
Pars reticularis
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Projections back to cortex and basal ganglia
Projections from cortex and basal ganglia

→ **Dopaminergic projection of substantia nigra**

→ **Thalamic and subthalamic projections**

→ **Cortical projection**

→ **Striatal projection**

→ **Corticostriatal projection**

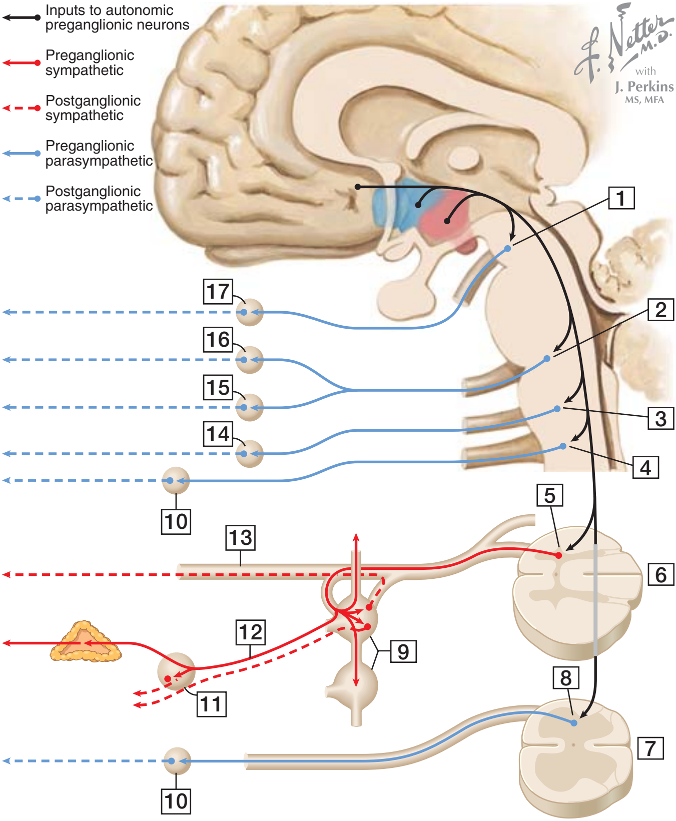
→ **Pallidal projection**

Connections of the Basal Ganglia

1. Caudate nucleus
2. Corticostriate projections
3. Striatopallidal projections
4. Putamen
5. Globus pallidus, external segment
6. Globus pallidus, internal segment
7. Fasciculus lenticularis
8. Ansa lenticularis
9. Raphe nuclei
10. Substantia nigra
11. Subthalamus
12. Centromedial (CM) thalamic nucleus
13. Ventral lateral (VL) thalamic nucleus
14. Ventral anterior (VA) thalamic nucleus

Comment: The basal ganglia consist of the striatum (caudate nucleus, putamen) and globus pallidus. The substantia nigra (SN) and subthalamic nucleus (STN), which are reciprocally connected with the basal ganglia, often are included as part of this system. Inputs to the basal ganglia derive from the cerebral cortex, thalamus (intralaminar nuclei), SN pars compacta (dopamine input), subthalamic nucleus, and rostral raphe nuclei (serotonergic input). Inputs from the STN reciprocally interconnect with the globus pallidus (GP). The internal segment of the GP projects to the thalamus (VL, VA, intralaminar nuclei), providing input to the cells of origin for the corticospinal system and other motor cortical output (to other brain stem UMN systems and to the basal ganglia). This allows the basal ganglia to assist the cortex in selecting wanted patterns of activity and suppressing unwanted patterns of activity. Damage to basal ganglia components often results in movement disorders. Damage to the dopamine neurons in SN pars compacta results in Parkinson’s disease, characterized by resting tremor, muscular rigidity, bradykinesia, and postural instability.

General Organization of the Autonomic Nervous System


Inputs to autonomic preganglionic neurons
Preganglionic sympathetic
Postganglionic sympathetic
Preganglionic parasympathetic
Postganglionic parasympathetic
F. Netter M.D.
with
J. Perkins
MS, MFA
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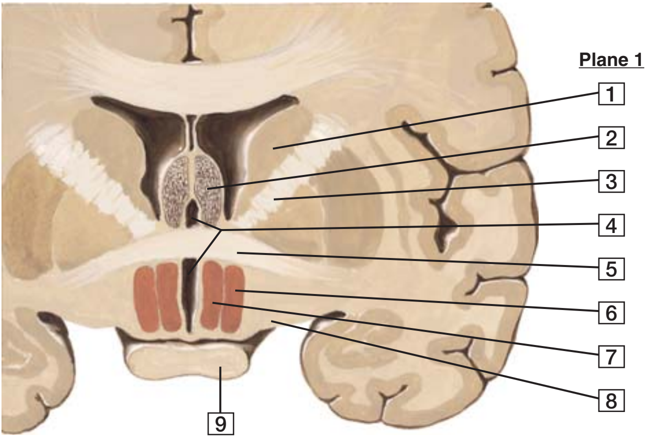
General Organization of the Autonomic Nervous System

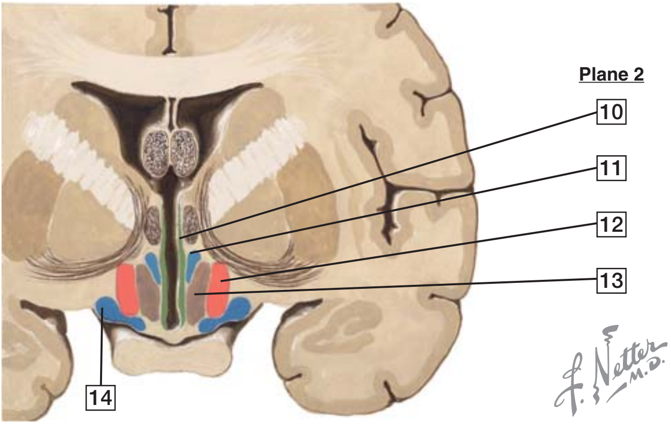
1. Nucleus of Edinger-Westphal
2. Superior salivatory nucleus
3. Inferior salivatory nucleus
4. Dorsal motor nucleus of CN X
5. Lateral horn (intermediolateral cell column)
6. Thoracic spinal cord (T1-L2)
7. Sacral spinal cord (S2-S4)
8. Intermediate gray
9. Sympathetic chain ganglion
10. Intramural ganglion
11. Collateral ganglion
12. Splanchnic nerve
13. Spinal nerve
14. Otic ganglion
15. Submandibular ganglion
16. Pterygopalatine ganglion
17. Ciliary ganglion

Comment: The autonomic nervous system is a two-neuron chain connecting preganglionic neurons through ganglia to visceral target organs (smooth muscle, cardiac muscle, secretory glands, metabolic organs, cells of the immune system). The sympathetic division (SNS) is a thoracolumbar (T1-L2) system arising from the intermediolateral cell column, sending preganglionic nerve fibers to sympathetic chain ganglia (paravertebral) and collateral ganglia (prevertebral). It is a fight-or-flight system reacting to emergencies or stressful situations. The parasympathetic division (PSNS) is a craniosacral system arising from cranial nerve nuclei of the brain stem (CNs III, VII, IX, X) acting through associated cranial nerve ganglia in or near the target organs, as well as from intermediomedial neurons in the sacral spinal cord (S2-S4) acting through intramural ganglia in or near the target organs. The PSNS is a homeostatic reparative system active during quiet, reparrative, or nonthreatening periods. Central connections from limbic forebrain structures, the hypothalamus, and brain stem nuclei regulate the SNS and PSNS outflow to the body, especially through connections to the preganglionic neurons in the intermediolateral cell column and dorsal motor nucleus of the vagus (via nucleus solitarius).

Sections through the Rostral Hypothalamus: Preoptic and Supraoptic Zones

Sections through hypothalamus I





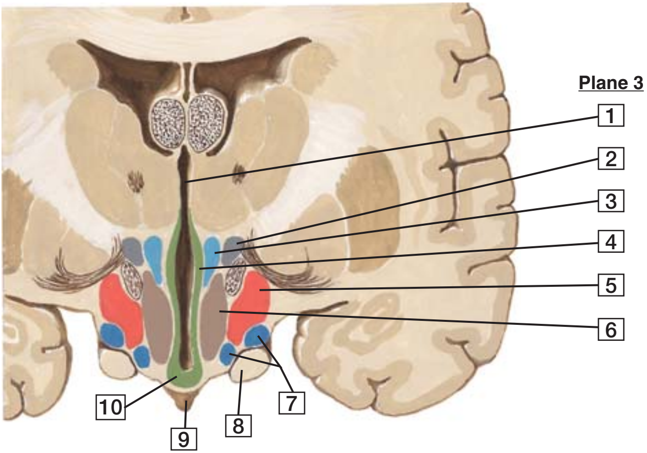
Sections through the Rostral Hypothalamus: Preoptic and Supraoptic Zones

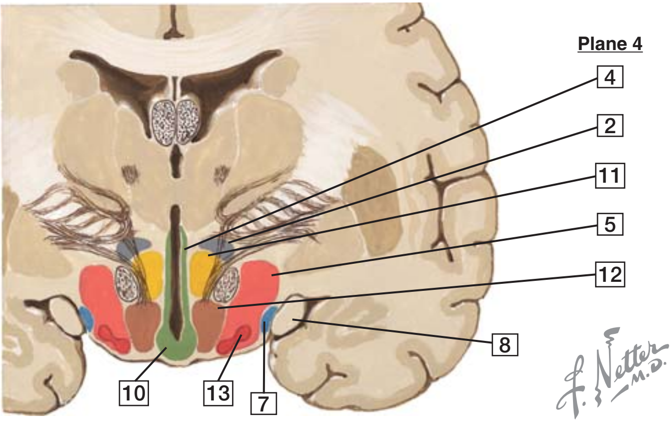
1. Head of the caudate nucleus
2. Column of the fornix
3. Anterior limb of the internal capsule
4. Third ventricle
5. Anterior commissure
6. Lateral preoptic area
7. Medial preoptic area
8. Substantia innominata
9. Optic chiasm
10. Periventricular nucleus
11. Paraventricular nucleus
12. Lateral hypothalamic area
13. Anterior hypothalamic area
14. Supraoptic nucleus

Comment: The major nuclei in the preoptic zone include the medial and lateral preoptic nuclei, involved in the sleep-wake cycle, thermoregulation, and other visceral functions. The organum vasculosum of the lamina terminalis (OVLT) is a circumventricular organ with no blood-brain barrier; neurons of the OVLT can release prostaglandin E2, acting on distant structures of the nervous system. The major nuclei of the supraoptic (anterior) zone include the supraoptic and paraventricular nuclei (cells of origin for posterior pituitary hormone synthesis, and for visceral autonomic regulation and anterior pituitary regulation), the anterior hypothalamic area (involved in autonomic regulation), and the lateral hypothalamic area (involved in feeding and drinking behavior). Some regions (paraventricular nucleus) have 20 or more chemically specific and anatomically specific subsets of neurons with discrete projections and visceral and neuroendocrine functions. These neuronal subgroups may be intermingled at a single locale.

Sections through the Midhypothalamus: Tuberal Zone

Sections through hypothalamus II





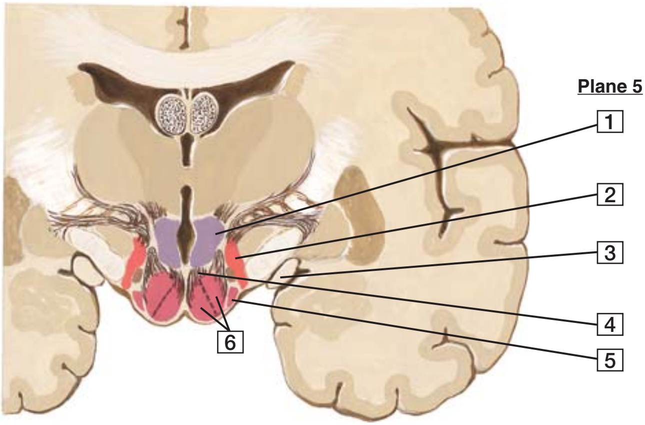
Sections through the Midhypothalamus: Tuberal Zone

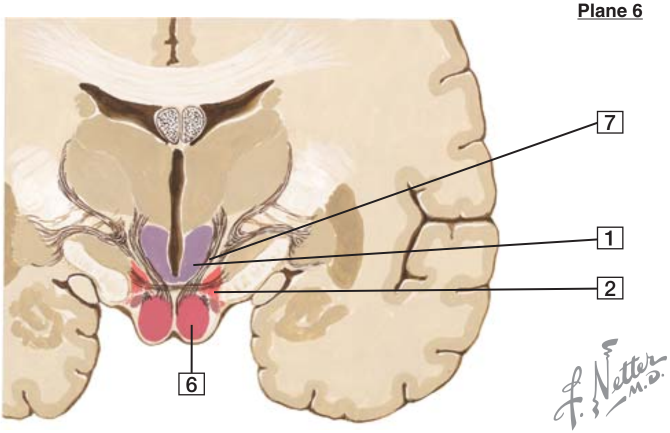
1. Third ventricle
2. Dorsal hypothalamic area
3. Paraventricular nucleus
4. Periventricular nucleus
5. Lateral hypothalamic area
6. Anterior hypothalamic area
7. Supraoptic nucleus
8. Optic tract
9. Infundibulum
10. Periventricular arcuate nucleus
11. Dorsomedial nucleus
12. Ventromedial nucleus
13. Tuberal nuclei

Comment: The major nuclei in the tuberal zone include the dorsomedial nucleus, ventromedial nucleus, periventricular nucleus, arcuate nucleus, periarcuate area (beta endorphin cells), tuberal nuclei, dorsal hypothalamic area, and lateral hypothalamic area. These neurons regulate a variety of visceral functions (feeding, drinking, reproductive behavior) and neurohormonal activities of the anterior pituitary gland. The median eminence extends from this region; axons from releasing-factor and inhibitory-factor neurons that control the release of anterior pituitary hormones end at the contact zone of the median eminence and release their factors into the hypophysial portal system, a local vascular system that bathes the cells of the anterior pituitary with high concentrations of those factors.

Sections through the Caudal Hypothalamus: Mammillary Zone

Sections through hypothalamus III


Plane 5

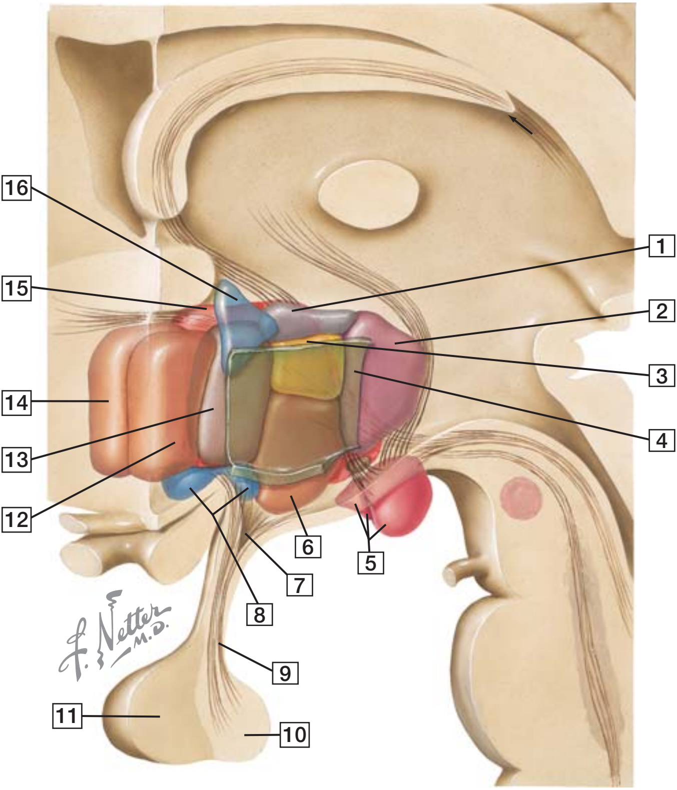

Plane 6

Sections through the Caudal Hypothalamus: Mammillary Zone

1. Posterior hypothalamic area
2. Lateral hypothalamic area
3. Optic tract
4. Principal mammillary fasciculus
5. Lateral mammillary nucleus
6. Medial and lateral portions of the medial mammillary nucleus
7. Mammillothalamic tract

Comment: The major nuclei in the mammillary zone include the medial and lateral mammillary nuclei (involved in limbic circuitry associated with short-term memory function), the posterior hypothalamic area (helps to regulate activity of the sympathetic nervous system), and the lateral hypothalamic area (involved in visceral hypothalamic functions). The lateral hypothalamic area extends throughout most of the hypothalamus and shows neuronal characteristics seen in the brain stem reticular formation.

Schematic Reconstruction of the Hypothalamus

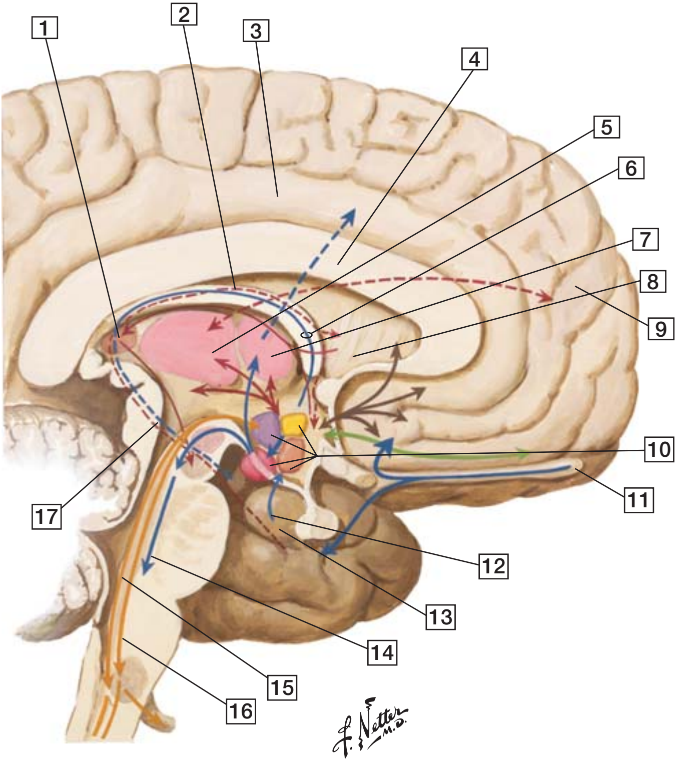


Schematic Reconstruction of the Hypothalamus

1. Dorsal hypothalamic area
2. Posterior hypothalamic area
3. Dorsomedial nucleus
4. Periventricular nucleus
5. Mammillary complex
6. Ventromedial nucleus
7. Tuberohypophysial tract
8. Supraoptic nucleus
9. Supraopticohypophysial tract
10. Posterior lobe of the pituitary gland
11. Anterior lobe of the pituitary gland
12. Medial preoptic nucleus
13. Anterior hypothalamic area
14. Lateral preoptic nucleus
15. Lateral hypothalamic area
16. Paraventricular nucleus

Comment: This schematic 3D reconstruction of the hypothalamus in sagittal section shows the nuclei, areas, and zones that occupy this small, compact region of the diencephalons. These regions are interconnected with limbic forebrain structures (via the fornix and median forebrain bundle); the anterior and posterior lobes of the pituitary gland (via the supraopticohypophysial tract and tuberohypophysial tract); brain stem and spinal cord autonomic structures (via the descending median forebrain bundle, dorsal longitudinal fasciculus, mammillotegmental tract, and descending connections from the paraventricular nucleus to preganglionic autonomic neurons); and the thalamus (via the mammillothalamic tract).

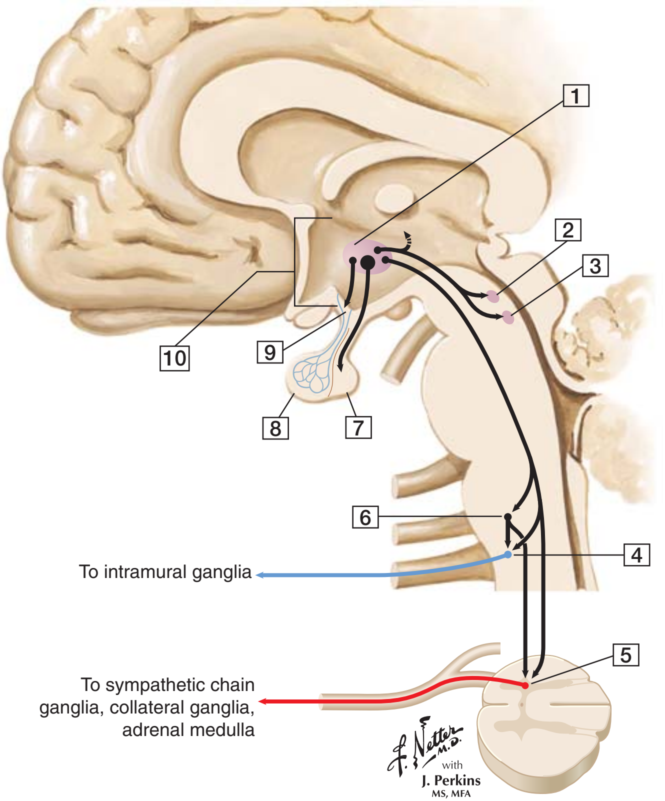
Afferent and Efferent Pathways Associated with the Hypothalamus



Afferent and Efferent Pathways Associated with the Hypothalamus

1. Habenula
2. Stria medullaris thalami
3. Cingulate gyrus
4. Corpus callosum
5. Medial dorsal thalamic nucleus
6. Fornix
7. Anterior thalamic nucleus
8. Septal nuclei
9. Prefrontal cortex
10. Hypothalamic nuclei
11. Olfactory bulb
12. Ventral amygdalofugal pathway
13. Amygdala
14. Mammillotegmental tract
15. Dorsal longitudinal fasciculus
16. Median forebrain bundle (descending)
17. Stria terminalis

Comment: Afferent and efferent connections of the hypothalamus are numerous and complex. Inputs derive from the cerebral cortex (prefrontal, orbitofrontal); thalamus (anterior nuclei); hippocampal formation and subiculum (via the fornix); amygdaloid nuclei (via the stria terminalis and ventral amygdalofugal pathway); habenula (via fasciculus retroflexus); retina (suprachiasmatic nucleus); and numerous brain stem structures via several polysynaptic autonomic pathways. Efferents from the hypothalamus distribute to the median eminence (from many nuclei); posterior pituitary gland (supraopticohypophysial tract); septal nuclei and anterior perforated substance (ascending median forebrain bundle); thalamus (mammillothalamic tract); and many brain stem autonomic nuclei and spinal cord preganglionic neurons (dorsal longitudinal fasciculus, descending median forebrain bundle, mammillotegmental tract, direct descending connections from the paraventricular nucleus).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To intramural ganglia
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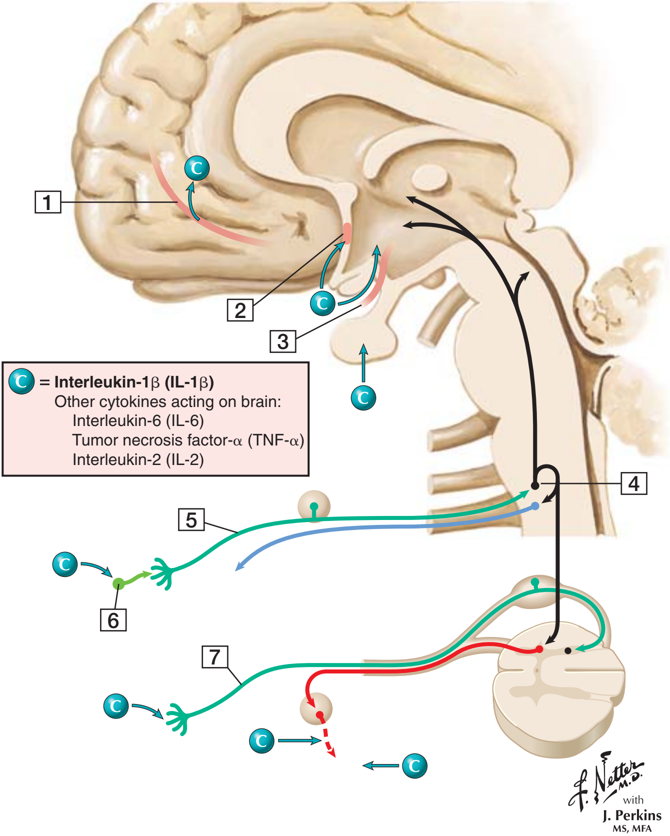
To sympathetic chain ganglia, collateral ganglia, adrenal medulla
F. Netter M.D.
with
J. Perkins
MS, MFA

Paraventricular Nucleus of the Hypothalamus

1. Paraventricular nucleus
2. Locus coeruleus
3. Parabrachial nuclei
4. Dorsal motor nucleus of CN X
5. Intermediolateral cell column in the lateral horn of T1-L2 spinal cord
6. Nucleus of the solitary tract (nucleus solitarius)
7. Posterior pituitary gland
8. Anterior pituitary gland
9. Hypophysial portal system in the median eminence
10. Hypothalamus

Comment: The paraventricular nucleus (PVN) is a small region of the hypothalamus adjacent to the third ventricle, containing a remarkable array of chemically specific neurons with multiple projections and regulatory roles related to neuroendocrine functions, visceral autonomic functions, and limbic functions. Magnocellular neurons send axons to the posterior pituitary, releasing oxytocin and vasopressin into the general circulation. Parvocellular neurons producing corticotropin-releasing factor (CRF) neurons (and some neurons producing vasopressin) send axons to the median eminence to release their hormones into the hypophysial portal system, influencing the release of ACTH. PVN parvocellular neurons send direct descending projections to preganglionic neurons in the intermediolateral cell column (sympathetic) and the dorsal motor nucleus of CN X and nucleus solitarius (parasympathetic). PVN neurons also project to several important limbic-related structures such as amygdaloid nuclei, parabrachial nuclei, and the locus coeruleus.

Mechanisms of Cytokine Influences on the Hypothalamus and Other Brain Regions and on Behavior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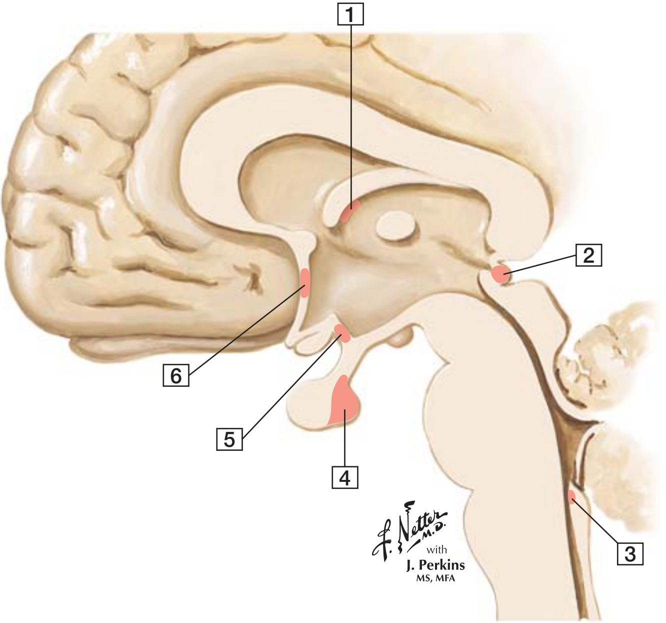
C
C = Interleukin-1β (IL-1β)
Other cytokines acting on brain:
Interleukin-6 (IL-6)
Tumor necrosis factor-α (TNF-α)
Interleukin-2 (IL-2)
F. Netter M.D.
with
J. Perkins
MS, MFA

Mechanisms of Cytokine Influences on the Hypothalamus and Other Brain Regions and on Behavior

1. Cerebral vasculature with blood-brain barrier (BBB)
2. Organum vasculosum of the lamina terminalis (OVLT)
3. Vasculature to the hypothalamus
4. Nucleus of the solitary tract
5. Vagal afferents in the viscera
6. Paraganglion cells associated with vagal afferents
7. Somatic afferents

Comment: Cytokines, including IL-1β, IL-6, TNF-α, and IL-2, can influence central neuronal activity and behavior. Cytokines such as IL-1β can access the brain by several mechanisms: (1) directly crossing the BBB into the brain, especially in cortical regions; (2) acting on neurons in circumventricular organs such as the OVLT, causing the release of mediators such as prostaglandin E2 (PGE2); (3) acting on vascular endothelial cells to release nitric oxide (NO), which acts in the central nervous system; (4) activating vagal afferents via stimulation of paraganglion cells, influencing their activities via projections to the nucleus of the solitary tract; (5) activating other afferent nerves in the periphery. IL-1β can evoke illness behavior, including fever, induction of slow-wave sleep, decreased appetite, lethargy, and classical illness symptoms. IL-1β also can influence autonomic and neuroendocrine regulation, as well as affective and cognitive function and behavior. IL-2 administration for immune-related cancer therapy had to be discontinued because it induced depression and suicidal behavior in some subjects.

Circumventricular Organs



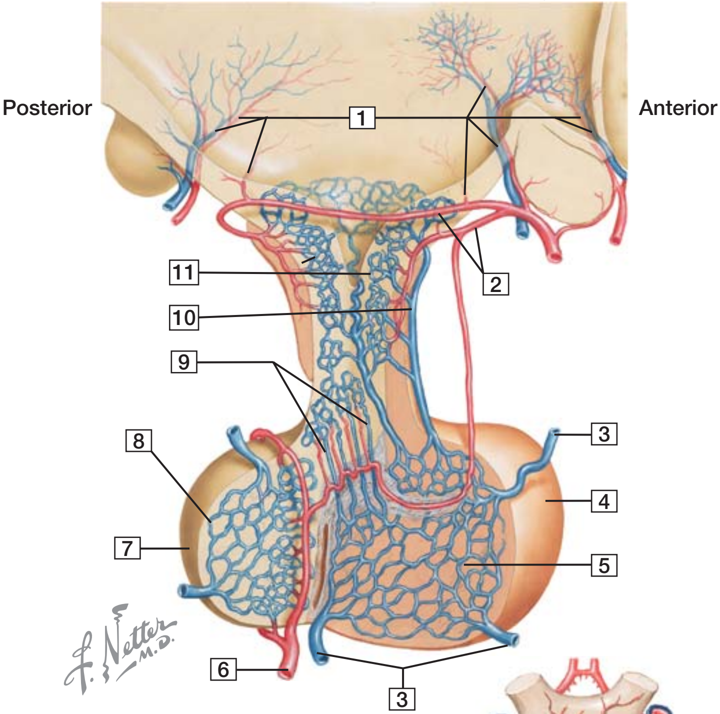
Circumventricular Organs

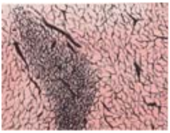
1. Subfornical organ
2. Pineal gland
3. Area postrema
4. Posterior pituitary gland
5. Median eminence
6. Organum vasculosum of the lamina terminalis (OVLT)

Comment: Circumventricular organs are known as “windows on the brain” that are devoid of a blood-brain barrier with the usual tight-junction endothelial appositions. The circumventricular organs have vasculature with fenestrated capillaries. Some circumventricular organs, such as the OVLT and area postrema, have neurons that project to the hypothalamus and other visceral structures. They also can release small molecule mediators such as prostaglandin E2 (PGE2), thus affecting target structures at a distance. The neurohypophysis is a site of release of oxytocin and vasopressin into the general circulation. The median eminence is the site of release of inhibitory factors and releasing factors for anterior pituitary hormones into the hypophysial portal system. The pineal gland is the site of synthesis and release of the hormone melatonin, influenced by postganglionic noradrenergic sympathetic input.

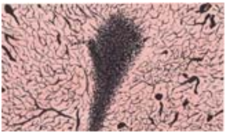
Hypophysial Portal Vasculature

Blood supply of hypothalamus and pituitary gland

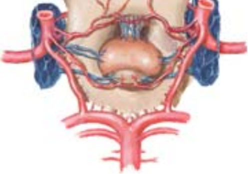




Section through paraventricular nucleus (*vessels injected*)



Similar section through supraoptic nucleus



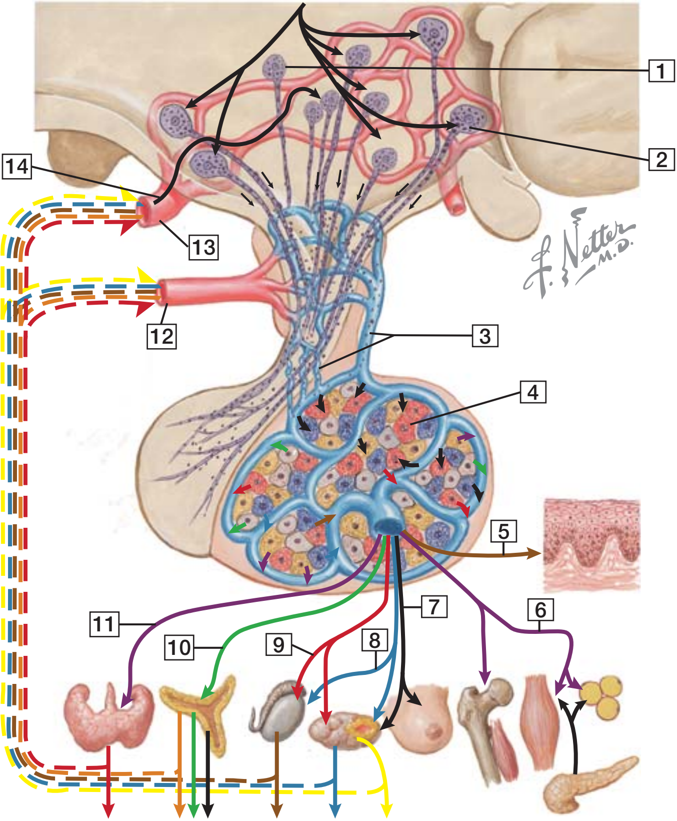
Inferior aspect

Hypophysial Portal Vasculature

1. Hypothalamic vessels
2. Superior hypophysial artery
3. Efferent vein to the cavernous sinus
4. Anterior lobe of the pituitary gland
5. Secondary plexus of hypophysial portal system
6. Inferior hypophysial artery
7. Posterior lobe of the pituitary gland
8. Capillary plexus of the infundibular process
9. Short hypophysial portal veins
10. Long hypophysial portal vein
11. Primary plexus of hypophysial portal system

Comment: The hypophysial portal vascular system derives from arterioles entering the median eminence at the base of the hypothalamus. The primary capillary plexus is the site where central axons release their inhibitory factors and releasing factors for anterior pituitary hormones (neurocrine secretions), which in turn are delivered in very high concentrations through the venules into the secondary capillary plexus to bathe cells that synthesize and secrete anterior pituitary hormones into the general circulation.

Regulation of the Anterior Pituitary Hormone Secretion

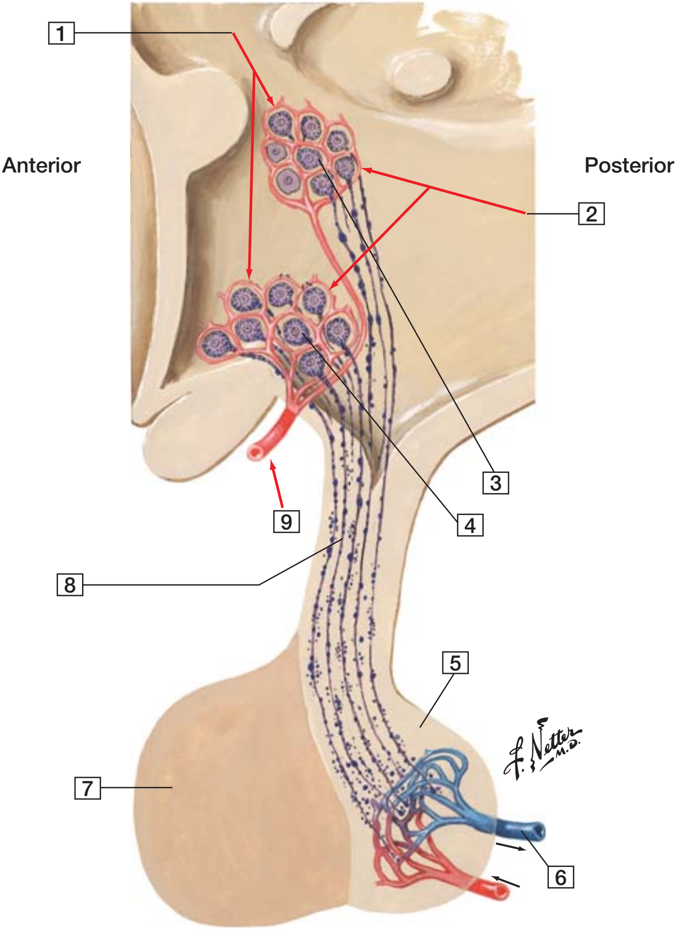


Regulation of the Anterior Pituitary Hormone Secretion

1. Neurons for releasing-factor and inhibitory-factor synthesis
2. Supraoptic nucleus
3. Hypophysial portal veins
4. Specific secretory cells of the anterior pituitary (pituicytes)
5. MSH (melanocyte-stimulating hormone)
6. STH (somatostatin, growth hormone)
7. LTH (prolactin)
8. LH (luteinizing hormone)
9. FSH (follicle-stimulating hormone)
10. ACTH (adrenal corticotropic hormone)
11. TSH (thyroid stimulating hormone)
12. Superior hypophysial artery
13. Hypothalamic artery
14. Blood-borne molecular influences on corticotropin-releasing factor (CRF) neurons

Comment: Neurons that synthesize releasing factors and inhibitory factors for regulation of anterior pituitary hormones are found in the hypothalamus and other brain regions. They send axons to the contact zone of the median eminence, where the axons release these factors into the hypophysial portal system to bathe the pituicytes in high concentrations of these factors. The anterior pituitary hormones act on peripheral target organs that include other endocrine structures (thyroid gland, adrenal gland, testes, ovaries, pancreas) and general target tissues (bone, muscle, fat cells, other tissues). Peripheral hormones from these target endocrine structures feed back to the hypothalamus and pituitary to regulate the subsequent production of releasing and inhibitory factors and anterior pituitary hormones. Thus, when there is an abundance of cortisol, synthesis and secretion of CRF and ACTH are decreased; this process occurs with endogenous regulation, as well as exogenous administration of pharmaceutical agents, such as corticosteroids.

Posterior Pituitary (Neurohypophysial) Hormones: Oxytocin and Vasopressin



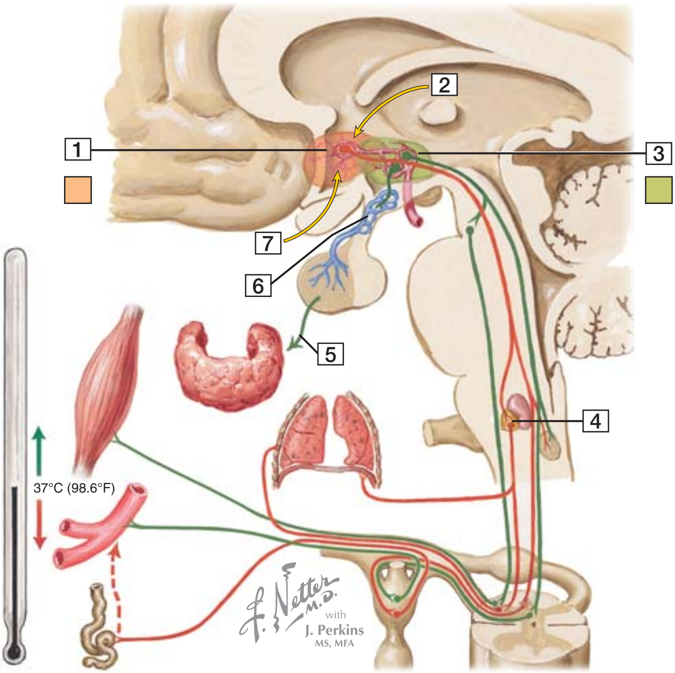
Posterior Pituitary (Neurohypophysial) Hormones: Oxytocin and Vasopressin

1. Forebrain pathways to supraoptic and paraventricular nuclei
2. Brain stem pathways to supraoptic and paraventricular nuclei
3. Paraventricular nucleus (PVN)
4. Supraoptic nucleus (SON)
5. Posterior lobe of the pituitary gland
6. Venous drainage of the posterior lobe
7. Anterior lobe of the pituitary gland
8. Neurohypophysial tract
9. Blood-borne signals reaching PVN and SON

Comment: The magnocellular neurons of the SON and PVN synthesize and secrete both oxytocin and arginine-vasopressin (antidiuretic hormone [ADH]), along with their neurophysin carrier proteins. These neurons send axons (supraopticohypophysial tract) into the posterior pituitary, where they terminate on fenestrated capillaries and secrete their hormones directly into the general circulation. Oxytocin neurons respond to estrogen and to afferent neuronal signals from suckling; oxytocin stimulates milk let-down and uterine contraction in pregnancy. Vasopressin neurons respond to changes in blood osmolarity, secreting vasopressin in the presence of high osmolarity; vasopressin causes the collecting tubules of the kidney to increase water resorption and prevents diuresis. Damage to the supraopticohypophysial tract (from pituitary stalk section) results in diabetes insipidus, with a loss of vasopressin secretion and production of large quantities of dilute urine, accompanied by polydipsia. Excessive vasopressin secretion (called *syndrome of inappropriate secretion of ADH* [SIADH]) may occur because of partial damage to the hypothalamus, a vasopressin-secreting tumor in the periphery, or pharmacologic agents. SIADH results in hypo-osmolar serum, hyponatremia, and high urine osmolarity.

See book 16.17

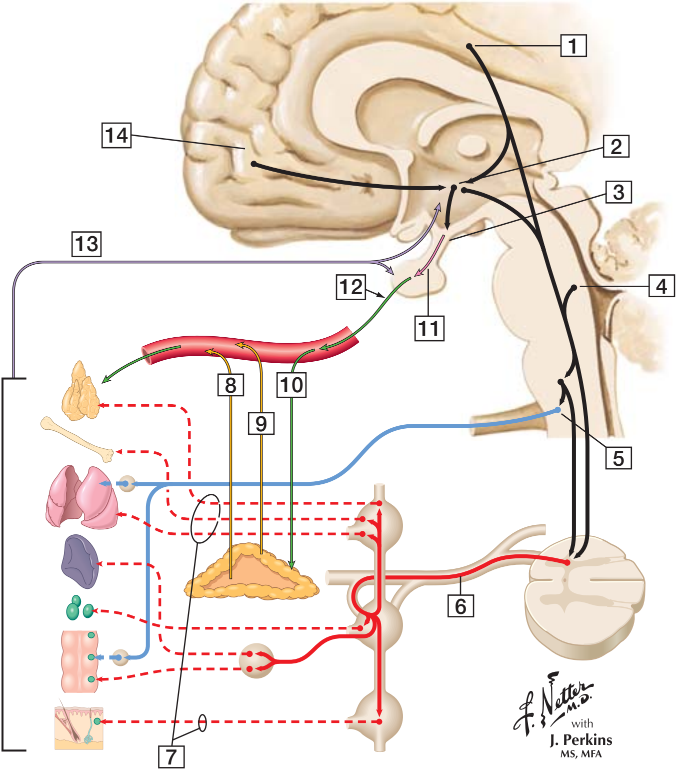
Hypothalamus and Thermoregulation



Hypothalamus and Thermoregulation

1. Thermoreception and regulation of heat loss
2. Afferent inputs from limbic forebrain structures
3. Hypothalamic region for conservation and production of heat
4. Respiratory centers
5. Thyrotropic hormone
6. Neurohumeral mechanism for increasing thyrotropic activity in the anterior pituitary to elevate metabolism
7. Action of inflammatory cytokines and pyrogens

Comment: The preoptic area of the hypothalamus contains heat-sensitive neurons and the posterior hypothalamic area contains cold-sensitive neurons. The preoptic area and anterior hypothalamic area initiate neuronal responses for heat dissipation (parasympathetic), and the posterior hypothalamic area initiates neuronal responses for heat generation (sympathetic). Neuronal pathways from limbic forebrain areas and brain stem nuclei can modulate the activity of these thermoregulatory neurons. The preoptic area also is responsive to pyrogens and the inflammatory cytokine, IL-1β; this area can generate a higher set point for temperature regulation, producing a disease-associated or inflammatory-associated fever. Extensive hypothalamic connections with the brain stem and spinal cord are used to initiate appropriate heat dissipation or heat generation responses. Appropriate behavioral responses also are initiated to optimize thermoregulation (e.g., seeking warmer or cooler location, using blankets for warmth or air conditioning for cooling).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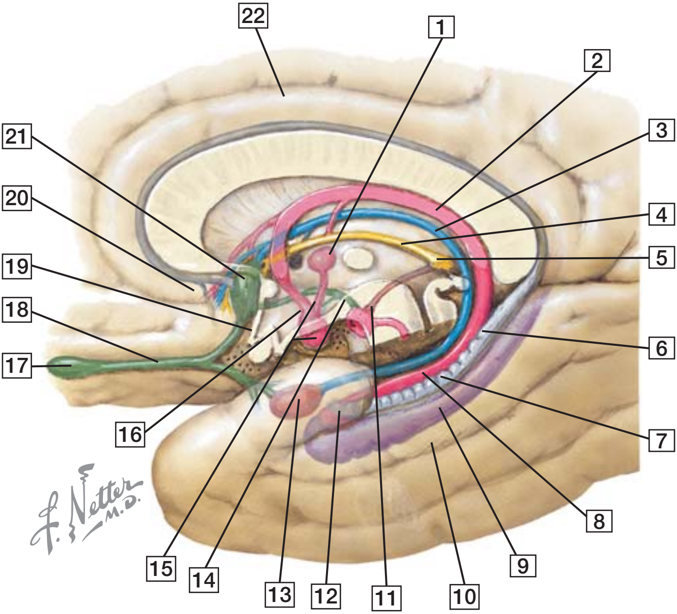
Neuroimmunomodulation

1. Limbic forebrain areas
2. Paraventricular nucleus of the hypothalamus
3. Median eminence
4. Brain stem nuclei
5. Nucleus of the solitary tract
6. Preganglionic sympathetic axons
7. Postganglionic sympathetic noradrenergic axons
8. Epinephrine and norepinephrine
9. Cortisol
10. ACTH
11. Site of action of releasing and inhibitory factors
12. Release of anterior pituitary hormones
13. Cytokine and inflammatory mediator feedback to the brain and pituitary
14. Cerebral cortex

Comment: Neurohormones from the anterior pituitary and sympathetic neuronal activity can influence a wide range of immune responses. Influences from the cerebral cortex, limbic forebrain structures, and brain stem provide the neuronal circuitry and substrate for behavioral influence, emotional responsiveness, chronic stressors, and positive complementary and behavioral influences that modulate immune responses through these neurohormones and the autonomic nervous system. Sympathetic postganglionic noradrenergic neurons directly contact immunocytes and vasculature in virtually all immune organs, including the parenchyma of bone marrow, thymus, spleen, lymph nodes, and mucosal- and skin-associated immune tissue. Virtually all anterior pituitary hormones, target organ hormones, and even some releasing-factor hormones (CRF) exert immunomodulatory influences. Cortisol from the hypothalamo-pituitary-adrenal (HPA) axis and norepinephrine and epinephrine from the sympathoadrenal system are particularly important in modulating immune reactivity as a component of the stress response, influencing primary immune cell proliferation and migration; antigen recognition, presentation, and processing; lymphocyte trafficking; generation of cell-mediated and humoral immune responses, including Th1 and Th2 activation; innate immune reactivity (natural killer cell activity, macrophage functions, others); immunoglobulin selection and production; and many other functions. Cytokines can modulate neural-immune interactions through induction of neurotransmitter secretion and modulation of immunocyte receptivity to neural signaling.

Anatomy of the Limbic Forebrain

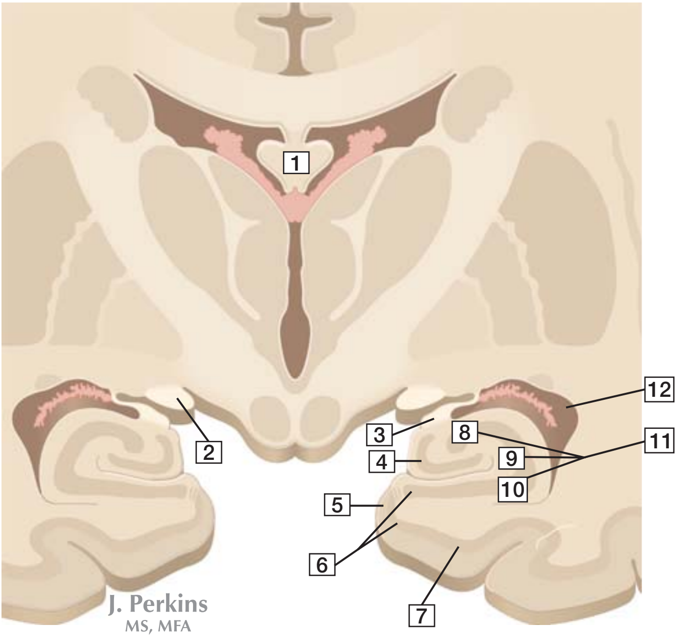
Major limbic forebrain structures



Anatomy of the Limbic Forebrain

1. Anterior thalamic nucleus
2. Fornix
3. Stria terminalis
4. Stria medullaris
5. Habenula
6. Gyrus fasciolaris
7. Dentate gyrus
8. Fimbria of the hippocampus
9. Hippocampus
10. Parahippocampal gyrus
11. Fasciculus retroflexus
12. Uncus (primary olfactory cortex)
13. Amygdaloid body (nuclei)
14. Median forebrain bundle
15. Mammillary body and mammillothalamic tract
16. Postcommissural fornix
17. Olfactory bulb
18. Olfactory tract
19. Lamina terminalis
20. Subcallosal area
21. Septal nuclei
22. Cingulate cortex

Comment: Structures of the limbic system are found in a rim that encircles the diencephalon. Two temporal lobe structures, the hippocampal formation with its pathway, the fornix, and the amygdala and its two pathways, the stria terminalis and ventral amygdalofugal pathway send projections into the hypothalamus and septal region in a C-shaped course. The septal nuclei project to the habenular nucleus via the stria medullaris thalami. The cingulate, prefrontal, orbitofrontal, entorhinal, and periamygdaloid cortical areas interconnect with subcortical and hippocampal components of the limbic forebrain and are often considered part of the limbic system. The limbic system is the substrate for regulation of emotional responsiveness and behavior, for individualized reactivity to sensory stimuli and internal stimuli, and for integrated memory tasks.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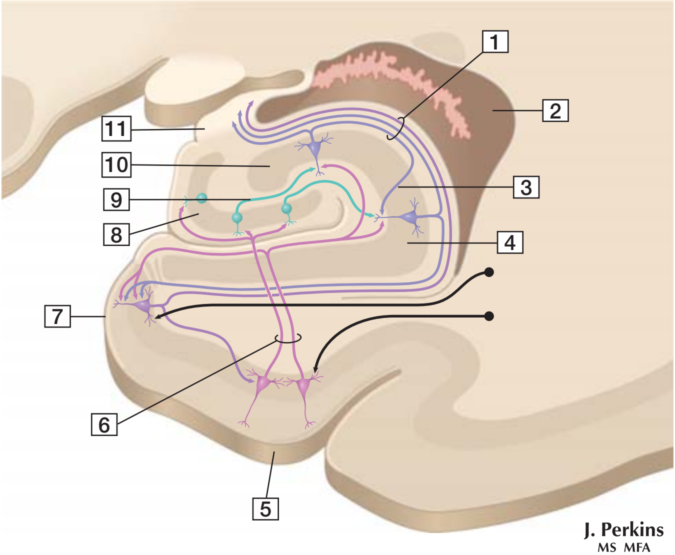
Hippocampal Formation: General Anatomy

1. Fornix
2. Optic tract
3. Fimbria
4. Dentate gyrus
5. Subiculum
6. Pyramidal cell layer of the subiculum
7. Pyramidal cell layer of the entorhinal cortex
8. CA3
9. CA2
10. CA1
11. CA regions of the hippocampal formation (pyramidal cells)
12. Temporal horn of the lateral ventricle

Comment: The hippocampal formation consists of the dentate gyrus, the hippocampus proper (with cornu ammonis [CA] regions), and the subiculum. These structures are connected with the adjacent entorhinal cortex. The hippocampus is named for its seahorse-like shape in the temporal lobe, bulging laterally into the temporal horn of the lateral ventricle. The hippocampus is divided into several zones of pyramidal cells, called *CA regions*, and reveals a three-layered cortical structure. Granule cells populate the dentate gyrus, which develops extensively postnatally, influenced by environmental stimuli. The hippocampal formation has extensive interconnections with cortical association areas and limbic forebrain structures (septal nuclei and cingulate cortex). The hippocampal formation is involved with the consolidation of short-term memory into long-term traces, in conjunction with extensive regions of the neocortex.

See book 16.26

Neuronal Connections of the Hippocampal Formation

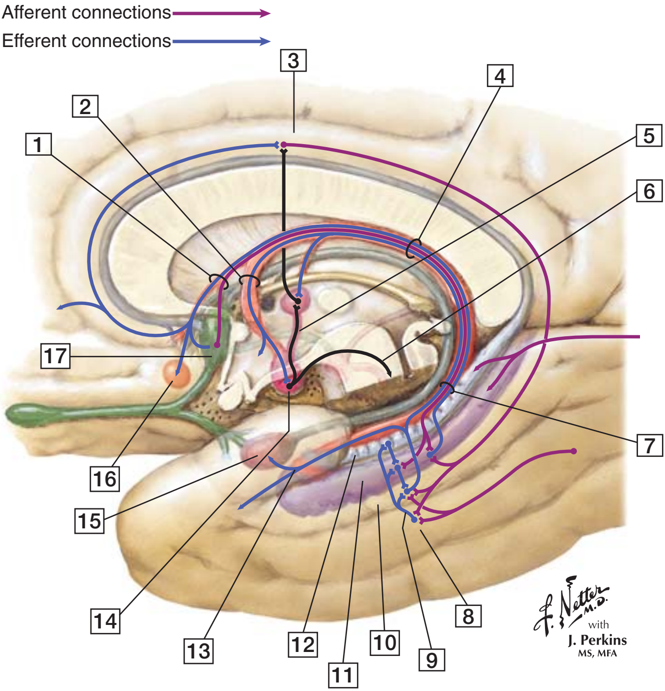


Neuronal Connections of the Hippocampal Formation

1.	Alveus	**7.**	Subiculum
2.	Temporal horn of the lateral ventricle	**8.**	Dentate gyrus
3.	Schaffer collaterals	**9.**	Mossy fiber
4.	CA1	**10.**	CA3
5.	Entorhinal cortex	**11.**	Fimbria
6.	Perforant pathway from the entorhinal cortex to dentate gyrus, CA1 and CA3, and the subiculum		

Comment: The hippocampal formation has an internal circuitry interconnected with the entorhinal cortex. Pyramidal neurons of the entorhinal cortex project to granule cell dendrites in the dentate gyrus; these granule cell axons (mossy fibers) synapse on pyramidal cell dendrites in CA3 of the hippocampus. Pyramidal cells in CA3 project to pyramidal cell dendrites in CA1 (Schaffer collaterals) and CA2. CA1 pyramidal axons project to pyramidal neurons in the subiculum. The subiculum sends axonal projections back to the pyramidal neurons of the entorhinal cortex, completing the circuit. Superimposed on this circuitry are other interconnections with association regions of the neocortex (from many sensory modalities) and from limbic forebrain structures. Neurons of CA1 and CA3 and the subiculum project axons into the fornix as efferent projections to target structures such as the hypothalamus (mammillary nuclei) and the septal nuclei. The subiculum also sends axons to the amygdala and to association areas of the temporal lobe. CA1 neurons are particularly vulnerable to ischemia, CA3 neurons are particularly vulnerable to high-dose corticosteroids (endogenous and exogenous), and both sets of pyramidal cells are particularly vulnerable to both ischemia and high steroid concentrations; damage results in severe loss of short-term memory.

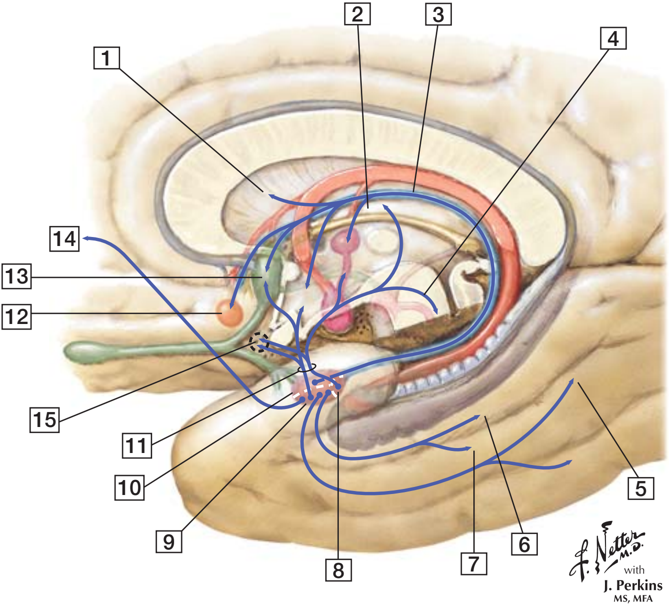
Major Afferent and Efferent Connections of the Hippocampal Formation



Major Afferent and Efferent Connections of the Hippocampal Formation

1. Precommissural fornix
2. Postcommissural fornix
3. Cingulate cortex
4. Fornix
5. Mammillothalamic tract
6. Mammillotegmental tract
7. Fimbria
8. Entorhinal cortex
9. Perforant pathway
10. Subiculum
11. CA regions of the hippocampus
12. Dentate gyrus
13. Efferents of the subiculum
14. Mammillary body (nuclei)
15. Amygdala
16. Nucleus accumbens
17. Septal nuclei

Comment: Pyramidal neurons in the subiculum and hippocampal regions CA1 and CA3 give rise to the efferent fornix. The subiculum projects to hypothalamic nuclei (especially mammillary nuclei) and thalamic nuclei via the postcommissural fornix. CA1 and CA3 neurons send axons to the septal nuclei, nucleus accumbens, the preoptic and anterior hypothalamic areas, the cingulate cortex, and association areas of the frontal lobe. Afferent cholinergic axons from septal nuclei traverse the fornix to supply the dentate gyrus and hippocampal CA regions; loss of this input may be associated with short-term memory dysfunction. Extensive inputs arrive in the hippocampal formation from sensory association cortices, prefrontal cortex, insular cortex, amygdaloid nuclei, and the olfactory bulb via the entorhinal cortex. The entorhinal cortex is fully integrated into the internal circuitry of the hippocampal formation. It is also reciprocally connected with the amygdala.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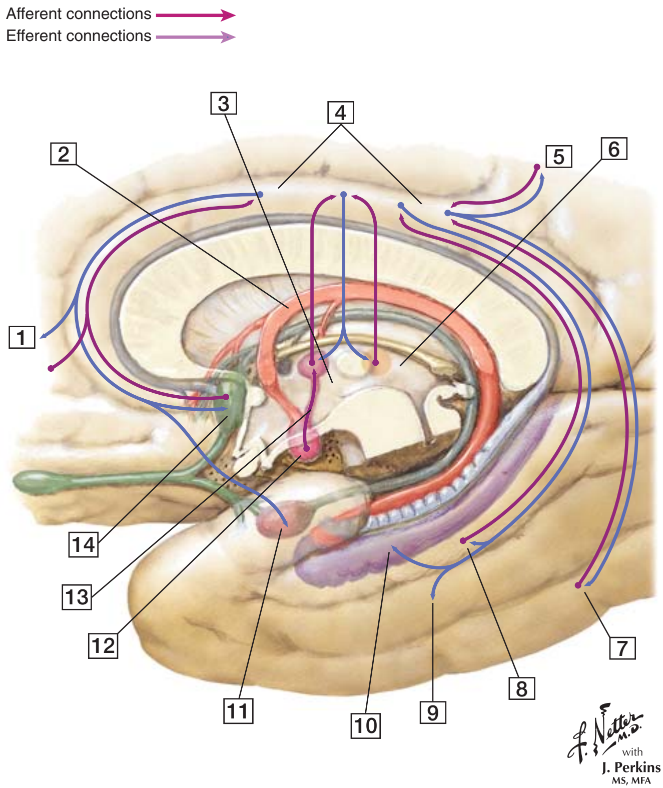
Major Efferent Connections of the Amygdala

1. Striatum
2. Bed nucleus of the stria terminalis
3. Stria terminalis
4. Amygdaloid projections to the brain stem
5. Amygdaloid projections to the cingulate cortex
6. Subiculum
7. Entorhinal cortex
8. Central nucleus of the amygdala
9. Basolateral nuclei of the amygdala
10. Corticomedial nuclei of the amygdala
11. Ventral amygdalofugal pathway
12. Nucleus accumbens
13. Septal nuclei
14. Amygdaloid projections to frontal and prefrontal cortex
15. Nucleus basalis in substantia innominate

Comment: Efferents from the corticomedial nuclei project through the stria terminalis and are directed mainly toward subcortical nuclei (septal nuclei, MD thalamic nucleus, hypothalamic nuclei, bed nucleus of the stria terminalis, nucleus accumbens, rostral striatum). Efferents from the basolateral nuclei project through the ventral amygdalofugal pathway to extensive cortical regions (frontal, cingulate, inferior temporal, entorhinal, subiculum) and subcortical limbic regions (hypothalamic nuclei, septal nuclei, cholinergic nucleus basalis in substantia innominata). The central amygdaloid nucleus receives inputs mainly from other amygdaloid nuclei and sends extensive efferents through the ventral amygdalofugal pathway to autonomic nuclei, monoaminergic neurons of the brain stem, midline thalamic nuclei, the bed nucleus of the stria terminalis, and cholinergic nucleus basalis. The amygdala is involved in providing emotional connotation of internal and external stimuli (individualized responses) and is particularly involved in fear responses. Bilateral damage to the amygdala abolishes fearful responses and results in other altered emotional responsiveness.

Major Connections of the Cingulate Cortex

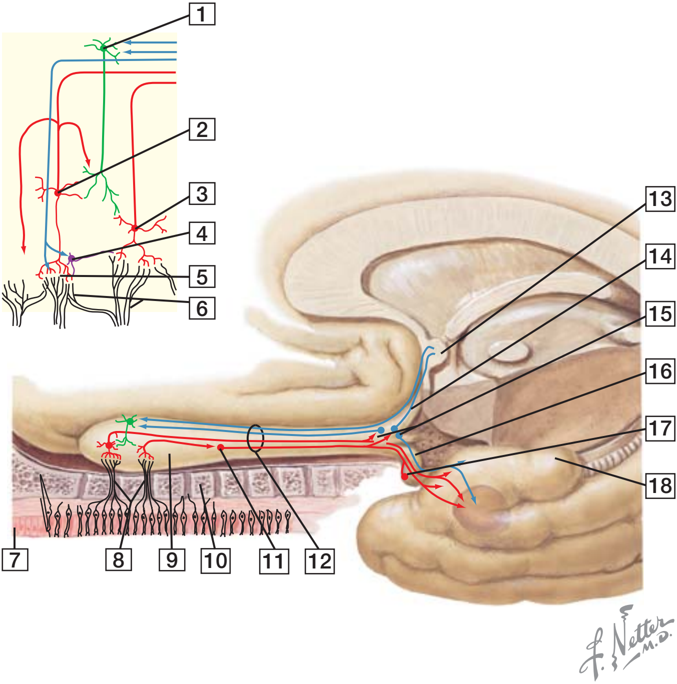


Major Connections of the Cingulate Cortex

1. Association areas of frontal cortex
2. Fornix
3. Anterior thalamic nucleus
4. Cingulate cortex
5. Association areas of parietal cortex
6. Medial dorsal thalamic nucleus
7. Association areas of temporal cortex
8. Subiculum
9. Entorhinal cortex
10. Hippocampus
11. Amygdala (basolateral nuclei)
12. Mammillary body (nuclei)
13. Mammillothalamic tract
14. Septal nuclei

Comment: The cingulate cortex is involved in the regulation of autonomic functions (respiratory, digestive, cardiovascular, pupillary); some somatic functions (motor tone, ongoing movements); and emotional responsiveness and behavior. Afferents arrive from association areas of the frontal, parietal, and temporal lobes; the subiculum; the septal nuclei; and thalamic nuclei (anterior, medial dorsal), all limbic-related structures. Efferents from the cingulate cortex project to association areas of the frontal, parietal, and temporal lobes, as well as to limbic forebrain regions (hippocampus, subiculum, entorhinal cortex, amygdaloid nuclei, and septal nuclei). These limbic forebrain structures provide regulation of autonomic and somatic activities through projections to the brain stem and spinal cord. Lesions of the cingulate cortex result in indifference to pain and other sensations that have emotional connotations and result in social indifference; these responses are similar to lesions of the orbitofrontal cortex.

Olfactory Pathways



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Olfactory Pathways

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1. Granule cell
2. Mitral cell
3. Tufted cell
4. Periglomerular cell
5. Glomerulus
6. Olfactory nerve fiber
7. Olfactory epithelium
8. Olfactory nerves
9. Olfactory bulb
10. Cribriform plate of the ethmoid bone
11. Anterior olfactory nucleus
12. Olfactory tract
13. Anterior commissure
14. Medial olfactory stria
15. Olfactory trigone and olfactory tubercle
16. Lateral olfactory stria
17. Lateral olfactory tract nucleus
18. Uncus (primary olfactory cortex)

Comment: Primary sensory olfactory axons from bipolar neurons of the olfactory epithelium pass through the cribriform plate and synapse in the olfactory glomeruli in the olfactory bulb. The olfactory nerve fibers synapse on dendrites of tufted and mitral cells, the secondary sensory neurons that give rise to the olfactory tract projections. Connections from serotonergic raphe neurons and noradrenergic locus coeruleus neurons modulate the excitability of tufted and mitral cells. The olfactory tract bypasses connections in the thalamus and projects directly to the anterior olfactory nucleus, nucleus accumbens, primary olfactory cortex (uncus), amygdaloid nuclei, periamygdaloid cortex, and lateral entorhinal cortex. The olfactory bulb interprets extensive information on odors (ability to detect several thousand separate odors) and sends the information directly to limbic regions involved in emotional interpretation, behavioral responsiveness, hypothalamic visceral functional activity for which odors are important, and to autonomic and neuroendocrine circuitry to regulate appropriate peripheral autonomic responses. Odors can influence food acquisition and appetite, territorial defense, relaxation or alertness responses, nausea, and many other important visceral responses. The olfactory cortex is interconnected with the orbitofrontal cortex, insular cortex, hippocampus, and lateral hypothalamus, further integrating olfaction with important visceral, emotional, and behavioral responsiveness.